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The Office of Drinking Water (ODW), U.S. Environmental Protection Agency has prepared a Drinking Water Criteria Document on Radon. This Criteria Document is an extensive review of the following topics:

- Physical and chemical properties of Radon
- Toxicokinetics and human exposure to Radon
- Health Effects of Radon in humans and animals
- Mechanisms of toxicity of Radon
- Quantification of toxicological effects of Radon

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CRITERIA DOCUMENT ON RADON

June, 1985

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I. SUMMARY

It has only been in the past sixty years that the health effects of inhaling radon have been recognized. It was discovered that the incidence of lung cancer was higher among hard rock and uranium miners exposed to radon. Only recently has the exposure to radon in other industrial locations, businesses and homes become known.

Radon can enter homes directly from the soil(and, hence, into both indoor and outdoor air) , through cracks and holes in the foundation, and from the drinking water. When transported in water, radon is released by such common household activities as washing clothes and dishes, baths, showers, flushing toilets, etc.

All three isotopes of radon (^{222}Rn, ^{220}Rn and ^{219}Rn) are chemically inert gases. Their half-lives are 3.8 days, 55 seconds and 4 seconds, respectively. Their main transport mechanisms are diffusion and carriage by convection currents. Due to short half-lives, the isotopes ^{220}Rn (thoron) and ^{219}Rn (actinon) contribute much less activity to indoor air than does ^{222}Rn (radon).

Since radon is chemically inert, it does not easily attach to the lung walls, although it can accumulate in lung tissue. The mechanism of human exposure involves decay of radon to its progeny (radioactive isotopes of polonium, lead and bismuth). These progeny (referred to historically as radon daughters) are electrically charged following radioactive decay and can attach themselves to atmospheric aerosols which subsequently deposit in the lung following inhalation.

Water samples containing radon can be collected, sealed and analyzed using a liquid scintillation technique. The method is quick, requires little preparation and involves equipment found in many laboratories.

Radon concentrations in water have been measured for a range of sources, including groundwater, tap water, rain water, oceans, rivers and thermal spas. The existing measurements of drinking water samples include about 4,000 individual measurements. These show positive measurements primarily for groundwater sources. The average radon concentration for all ground and surface water public drinking water supplies in the United States is in the range of 50 to 300 pCi/L.

As mentioned earlier, several household activities can lead to the release of radon into indoor air. Clearly, the radon concentration will be higher near the source, such as in a bathroom during a shower. The range of resulting radon air concentrations throughout the house can vary widely. On the average, however, 1 pCi of Rn/L of indoor air results from 10,000 pCi Rn/L of drinking water.

The activity of the radon progeny that deposit in the lung depends on several factors which are included in mathematical models. Once the radon progeny have been deposited within the bronchial passages, they tend to be removed by the mucociliary blanket, which carries them towards the trachea and, ultimately, the gastrointestinal (GI) tract. Decay of the progeny is sufficiently rapid to allow for almost complete decay during this transport. The dose per unit cumulative exposure for bronchial epithelium values extrapolated by calculated models is in the range of 0.5 to 0.8 rad/WLM. The working level month (WLM) is any combination of radon progeny in one liter of air which will result in the emission of 1.3×10^5 MeV of potential alpha energy.

The data that are available for estimating the risk due to ingested radon are skimpy. It is estimated, however, that the risk from ingested radon (not including the radon progeny in the water) is 5 to 500 times less than the risk due to the inhalation pathway.

Epidemiology data for occupational exposures to airborne radon progeny exist from the study of hard rock and uranium miners. The exposure estimates for these miners are usually crude; thus, risk factors can vary as much as an order of magnitude or more. The range of risk values used here is from 1.5 to 50 cancers/WLM/year/million people exposed.

Uncertainties in the effective dose and risk estimates include, among others, those due to analytical measurement uncertainties, sampling errors, variations in exposure level, non-equilibrium between airborne radon and progeny, variations in aerosol size distribution, dose modeling, uncertainties in epidemiology data, lack of knowledge about the critical cell affected, relative effectiveness of different radon progeny in causing cancers and variations in latent and plateau periods. Estimating these and other contributions suggests an overall uncertainty in the range of a factor of four to five.

The relationship between concentration of radon in drinking water to that in indoor air depends on many factors including air turnover rate, type of house, number of floors, type and frequency of water use, outdoor wind and humidity. As an average the ratio of indoor air concentration (the pCi per liter of air) to water concentration (the pCi per liter of water) is (0.17 to 3.5) X 10^{-4}.

Using data from the International Commission on Radiological Protection, Publication 32 (ICRP 1981), the effective dose equivalent per drinking water concentration for radon is in the range of 0.8 x 10^{-3} to 20 x 10^{-3} mrem/year/pCi/L. The risk of lung cancer is in the range of 2 x 10^{-8} to 60 x 10^{-8} excess lung cancer cases/pCi/L. The uncertainty in mean values for these risk estimates is a factor of four or five derived by Baseyian uncertainty analysis.

Radiological conversion factors are presented in Table I-1.

Table I-1 Radiological Conversion Factors

1 Becquerel (Bq) = 1 disintegration per second = 2.7 x 10^{-11} Curies.

1 Curie (Ci) = 3.7 x 10^{10} disintegrations per second.

1 Microcurie (μCi) = 10^{-6} Ci = 3.7 x 10^{4} disintegrations per second.

1 Picocurie (pCi) = 10^{-12} Ci = 3.7 x 10^{-2} disintegrations per second.

1 rad = 100 ergs per gram, deposited by radiation in matter.

1 Gray (Gy) = 100 rads = 10^{4} ergs/gram = 1 joule/kilogram.

rem = product of absorbed dose (rads) and the appropriate quality factor (QF).

1 Sievert (Sv) = 100 rems = 1 Gy times the QF.

1 Working Level (WL) = any combination of radon progeny yielding a potential alpha energy of 1.3 x 10^{5} MeV, if all progeny complete their chain of decays, per liter of air. This corresponds to 100 pCi/liter of each radon progeny. In S.I. units, 1 WL equals 2.08 x 10^{-5} joules per cubic meter of air.

1 Working Level Month (WLM) = exposure to 1 WL for a total of 170 hours. Continuous exposure to 1 WL for a year results in 51.53 WLM/year.

1 Bq/m^3 = 2.7 x 10^{-2} pCi/liter.

II. PHYSICAL AND RADIOLOGICAL BEHAVIOR OF RADON

A. Introduction

The present report focuses entirely on the most prevalent isotope of radon, ^{222}Rn. In actuality, there are at least three isotopes of radon found in natural sources. These are ^{219}Rn, ^{220}Rn and ^{222}Rn, typically known by their historical names of actinon, thoron and radon, respectively. These isotopes are the products of radioactive decay chains that begin with some element other than the radon isotope and cascade past the radon isotope to a stable isotope of Pb. Partial summaries of the three associated decay chains for ^{219}Rn, ^{220}Rn and ^{222}Rn are displayed in Figures II-1, II-2 and II-3, respectively. The primary source of ^{222}Rn is the presence of ^{226}Ra in soil and rocks.

All three isotopes of radon are chemically inert gases, falling within the classification of noble gases. [For a complete review of their behavior, the reader is referred to the classic review by Evans (1969) or to the recent summary provided in NCRP 19846.] Being chemically inert, the gases usually do not enter into chemical reactions, which allows them to diffuse freely through materials. If, however, their radium precursors are present in solidified matrices of material, such as granite, the atoms of radon often are trapped physically within the matrix and undergo only slow diffusion. This velocity of diffusion determines the extent

Radionuclide	Historical name	Half-life	Major radiation energies and intensities					
			α		β		γ	
			MeV	%	MeV	%	MeV	%
$^{235}_{92}\mathrm{U}$	Actinouranium	7.1 × 10⁸y	4.37 4.40 4.58c	18 57 8	———		0.143 0.185 0.204	11 54 5
$^{231}_{90}\mathrm{Th}$	Uranium Y	25.5h	———		0.140 0.220 0.305	45 15 40	0.026 0.084	2 10
$^{231}_{91}\mathrm{Pa}$	Protoactinium	3.25 × 10⁴y	4.95 5.01 5.02	22 24 23	———		0.027 0.290	6 6
$^{227}_{89}\mathrm{Ac}$	Actinium	21.6y	4.86c 4.95c	0.18 1.2	0.043	~99	0.070	0.08
98.6% 1.4%								
$^{227}_{90}\mathrm{Th}$	Radioactinium	18.2d	5.76 5.98 6.04	21 24 23	———		0.050 0.237 0.310	8 15 8
$^{223}_{87}\mathrm{Fr}$	Actinium K	22m	5.44	~0.005	1.15	~100	0.050 0.080 0.234	40 13 4
$^{223}_{88}\mathrm{Ra}$	Actinium X	11.43d	5.61 5.71 5.75	26 54 9	———		0.149 0.270 0.33	10 10 6
$^{219}_{86}\mathrm{Rn}$	Emanation Actinon (An)	4.0s	6.42 6.55 6.82	8 11 81	———		0.272 0.401	9 5
$^{215}_{84}\mathrm{Po}$	Actinium A	1.78ms	7.38	~100	0.74	~.00023	———	
~100 .00023%								
$^{211}_{82}\mathrm{Pb}$	Actinium B	36.1m	———		0.29 0.56 1.39	1.4 9.4 87.5	0.405 0.427 0.832	3.4 1.8 3.4
$^{215}_{85}\mathrm{At}$	Astatine	0.1ms	8.01	~100	———		———	
$^{211}_{83}\mathrm{Bi}$	Actinium C	2.15m	6.28 6.62	16 84	0.60	0.28	0.351	14
0.28% 99.7%								
$^{211}_{84}\mathrm{Po}$	Actinium C′	0.52s	7.45	99	———		0.570 0.90	0.5 0.5
$^{207}_{81}\mathrm{Tl}$	**Actinium C″**	**4.79m**	———		**1.44**	**99.8**	**0.897**	**0.16**
$^{207}_{82}\mathrm{Pb}$	Actinium D	Stable	———		———		———	

Figure II-1 ^{235}U Decay Chain Characteristics

Radionuclide	Historical name	Half-life	Major radiation energies and intensities					
			α		β		γ	
			MeV	%	MeV	%	MeV	%
$^{228}_{90}\mathrm{Th}$	Radiothorium	1.913 a	5.34 5.43	27 73			0.084 0.216	1.2 0.3
$^{224}_{88}\mathrm{Ra}$	Thorium X	3.66 d	545 5.68	6 94			0.241	3.9
$^{220}_{86}\mathrm{Rn}$	Emanation Thoron (Tn)	55 s	6.29	100			0.55	0.1
$^{216}_{84}\mathrm{Po}$	Thorium A	0.15 s	6.78	100				
$^{212}_{82}\mathrm{Pb}$	Thorium B	10.64 h			0.331 0.569	83 12	0.239 0.300	43 3.2
$^{212}_{83}\mathrm{Bi}$	Thorium C	60.6 min	6.05 6.09	25 10	1.55 2.26	5 55	0.040 0.727 1.620	1.1 11.8 2.8
64% 36%								
$^{212}_{84}\mathrm{Po}$	Thorium C'	304 ns	8.78	100				
$^{208}_{81}\mathrm{Tl}$	Thorium C"	3.05 min			1.28 1.52 1.80	23 22 51	0.511 0.583 0.860 2.614	23 86 12 100
$^{208}_{82}\mathrm{Pb}$	Thorium D	Stable						

Figure II-2 ^{232}Th Decay Chain Characteristics

Radionuclide	Historical name	Half-life	Major radiation energies and intensities					
			α		β		γ	
			MeV	%	MeV	%	MeV	%
$^{226}_{88}\mathrm{Ra}$	Radium	$1.6\ 10^3$ a	4.60 4.78	6 94			0.186	3.3
$^{222}_{86}\mathrm{Rn}$	Emanation Radon (Rn)	3.823 d	5.49	100				
$^{218}_{84}\mathrm{Po}$	Radium A	3.05 min	6.00	100				
99.98% | 0.02%								
$^{214}_{82}\mathrm{Pb}$	Radium B	26.8 min			0.67 0.73 1.02	48 42 6	0.295 0.352	19 37
$^{218}_{85}\mathrm{At}$	Astatine	~2 s	6.65 6.69 6.76	6 90 3.6	?	~0.1		
$^{214}_{83}\mathrm{Bi}$	Radium C	19.7 min	5.45 5.51	0.012 0.008	1.0 1.51 3.26	23 40 19	0.609 1.12 1.764	46 15 16
99.98% | 0.02%								
$^{214}_{84}\mathrm{Po}$	Radium C′	164 μs	7.69	100				
$^{210}_{81}\mathrm{Tl}$	Radium C″	1.3 min			1.3 1.9 2.3	25 56 19	0.296 0.795 1.31	80 100 21
$^{210}_{82}\mathrm{Pb}$	Radium D	22.3 a			0.015 0.061	81 19	0.047	4
$^{210}_{83}\mathrm{Bi}$	Radium E	5.01 d			1.161	100		
100% | 0.0001%								
$^{210}_{84}\mathrm{Po}$	Radium F	138.4 d	5.305	100				
$^{206}_{81}\mathrm{Tl}$	Radium E″	4.2 min			1.53	100		
$^{206}_{82}\mathrm{Pb}$	Radium G	Stable						

Figure II-3 ^{226}Ra Decay Chain Characteristics to which the radon is able to reach any exposed surfaces and emanate into the air or into bodies of ground or surface water.

The radioactive half-lives of the radon isotopes (defined as the amount of time necessary for one-half of an initial number of radon atoms to undergo decay) differ greatly. The isotope ^{219}Rn has a half-life of 3.96 seconds, ^{220}Rn has a half-life of 55 seconds, and ^{222}Rn has a half-life of 3.823 days (Lederer 1967 or HEW 1970). Because of the very short half-lives of ^{219}Rn and ^{220}Rn, these isotopes rarely are able to diffuse from their sites of production and enter the air. Figure II-4 displays the relative survival fractions for the three isotopes of radon as a function of depth of a source in porous soil (Crawford 1981), assuming a diffusion velocity of 4 cm/hr.

Given equal source terms (number of atoms of the radon isotope formed per gram of soil), it may be seen that the quantity of ^{219}Rn or ^{220}Rn that survives migration through any distance of soil is a small fraction of the amount of ^{222}Rn surviving an equal migration distance. For purposes of calculation, a velocity of 4 cm/hour was chosen as a typical diffusion velocity of inert gases through porous soil (Tanner 1978). It will be noted from Figure II-4 that ^{222}Rn may travel many tens of meters through typical soils, while ^{219}Rn and ^{220}Rn are restricted to migrations of a few millimeters and a few centimeters, respectively, when movement is confined to diffusion. The assumption of equal source terms is usually

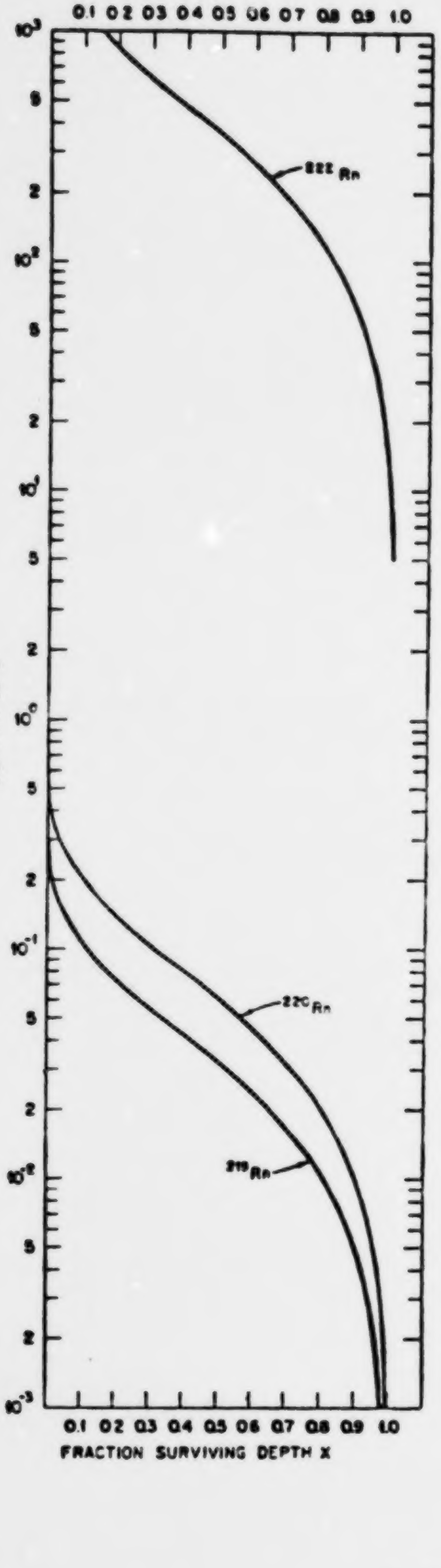

Figure II-4 Attenuation of the Isotopes of Radon During Migration Through Soil

Adapted from Tanner 1978.

valid for ^{220}Rn and ^{222}Rn, since normal soils contain approximately 1 pCi/g of both ^{232}Th and ^{238}U (Myrick 1981). The actinium chain, however, usually is present in an activity which is only 5% of the activity from the ^{238}U chain. This low abundance lends even more credence to the assumption that ^{219}Rn exposures should be much lower than those to ^{222}Rn.

The net result of these considerations is that ^{222}Rn is much more likely than either ^{219}Rn or ^{220}Rn to provide human exposures to radiation. This conclusion is supported by the lack of measureable ^{219}Rn in air in the United States despite sensitive measurements (Myrick 1979) and by the much lower estimates of ^{220}Rn exposures published by UNSCEAR (see Table II-1 and compare to Table II-2 both taken from UNSEAR 1982). The contribution from ^{220}Rn to the world population is a factor of five below that from ^{222}Rn, with the ^{219}Rn contribution being below measureable limits except at a few special industrial locations (Crawford 1981). Pertinent physical and chemical properties of ^{222}Rn are displayed in Tables II-3 and II-4.

As may be seen in Figure II-3, ^{222}Rn decays through alpha emission to ^{218}Po (Radium A), with the decay chain continuing through several more elements and terminating at the stable isotope, of lead ^{206}Pb. Since the radiological hazard from inhaled ^{222}Rn and progeny resides almost entirely in the progeny and not in the ^{222}Rn itself (since the ^{222}Rn is

Table II-1 Estimated Equilibrium Concentration of ^{220}Radon and Annual Effective Dose Equivalents Arising From Inhalation

	Populations in temperate regions	Global population
Outdoor exposure		
Equilibrium equivalent concentration (Bq m^{-3})	0.2	0.2
Annual effective dose equivalent (mSV)	0.02	0.02
Indoor exposure		
Equilibrium equivalent concentration (Bq m^{-3})	0.7	
Annual effective dose equivalent (mSV)	0.20	0.15
Total annual effective dose equivalent (mSV)	0.22	0.17

Source: UNSEAR 1977

Table II-2 Estimated Equilibrium Equivalent Concentration of ^{222}Rn and Annual Effective Dose Equivalents Arising From Inhalation

	Populations in temperate regions	Global population
Outdoor exposure		
Equilibrium equivalent concentration (Bq m^{-3})	1.8	1.8
Range of mean annual values (Bq m^{-3})	0.1-10	0.1-10
Average annual effective dose equivalent (mSV)	0.06	0.06
Range of mean annual values (mSV)	0.003-0.3	0.003-0.6
Indoor exposure		
Equilibrium equivalent concentration (Bq m^{-3})	15	
Range of mean values in various countries (Bq m^{-3})	5-60	
Average annual effective dose equivalent (mSV)	0.92	0.7
Range of mean values in various countries (mSV)	0.3-3.7	
Total average annual effective dose equivalent (mSV)	1.0	0.8

Source: UNSEAR 1977

Table II-3 Properties of ^{222}Rn

Property		Value
Volume of 27.03 pCi of ^{222}Rn at NTP[a]		1.6×10^{-20} m^3
Boiling point		- 61.8°C
Melting point		- 71°C
Vapor pressure at	- 144.0°C	0.13 kPa
	- 126.3°C	1.3 kPa
	- 111.3°C	5.3 kPa
	- 99.0°C	13 kPa
	- 71.0°C	53 kPa
	- 61.8°C	100 kPa
Density at NTP		9.96 kg m^{-3}
Coefficients of solubility[b] at atmospheric pressure:		
in water at:	0°C	0.507
	10°C	0.340
	20°C	0.250
	30°C	0.195
	37°C	0.167
	50°C	0.138
	75°C	0.114
	100°C	0.106

continued -

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[a] NTP = Normal temperature and pressure.

[b] The solubility coefficient or partition coefficient in water is defined as the ratio, C_w/C_a, where C_w and C_a are the radon concentrations in water and air, respectively.

Table II-3 - continued

Property	Value	
	at 18°C	at 0°C
in: glycerine	0.21	-
aniline	3.80	4.45
absolute alcohol	6.17	8.28
acetone	6.30	7.99
ethyl acetate	7.35	9.41
petroleum (liquid paraffin)	9.20	12.6
xylene	12.75	-
benzene	12.82	-
toulene	13.24	18.4
chloroform	15.08	20.5
ether	15.08	20.09
hexane	16.56	23.4
carbon bisulfide	23.14	33.4
olive oil	29.00	-

Adapted from UNSCEAR (1982).

Table II-4 Characteristics of ^{222}Rn and its "Short-Lived" Progeny

Nuclide	T min	No. atoms/ 100 pCi	Decay mode	Alpha energy (MeV)	Ultimate decay (MeV-alpha)	WL
^{222}Rn	5500	(1.75 E+6)	(alpha)	(5.49)	(3.33 E+7)	--
^{218}Po	3.05	9.77 E+2	alpha	6.0	1.34 E+4	0.10
^{214}Pb	26.8	8.58 E+3	beta	--	6.60 E+4	0.52
^{214}Bi	19.6	6.28 E+3	beta	--	4.83 E+4	0.38
^{214}Po	2.7 E-6	8.8 E-4	alpha	7.69	6.7 E-3	0
Total sum of "Ultimate Alpha Energy"					1.28 E+5	1.00

Values (in parentheses) are not considered in computation of Working Level.

Table adapted from information in Evans 1969.

chemically inert and does not attach to the lung walls), concern is directed toward the concentration of ^{222}Rn progeny in the air. These progeny are produced in a charged state due to the decays which occur. As a result of this charge, these progeny (or daughter) atoms attach to the surface of atmospheric aerosols which may subsequently deposit in the lung following inhalation. These deposited atoms (as well as the unattached progeny) then contribute most of the dose delivered to the lung.

The attachment of the radon progeny to aerosols is governed by the number of aerosol particles in the air and the distribution of particle diameters (which determines the surface area available for attachment). Most studies of the size distribution of these aerosol particles indicate that the distribution of partical diameters is roughly log normal, with a geometric standard deviation of 2.5 to 3.0 (Hess et al. 1985). These results are approximated by the data of Soilleux (1970), Bergamini and Palmas (1973), Schumann (1963), Shapiro (1956) and Jacobi (1962). For purposes of calculation, the UNSCEAR report (1982) employed an Activity Median Aerodynamic Diameter (AMAD) of 0.05 micrometer and a geometric standard deviation of 3 for normal room air. The density of aerosol particles in air depends upon the location, with Jacobi (1964) basing attachment calculations on an assumed "normal room air" concentration of 10,000/cc, and with Haque and Collinson (1967) assuming 30,000/cc.

Results of dose calculations indicate that the lung dose is a function of the extent to which the radon progeny are attached to the aerosols. Raabe (1969) has published a theoretical development of this degree of attachment which agrees well with experimental data in air aged for more than 2 hours. According to this formulation, the equilibrium fraction of progeny atoms, f, that are not attached to aerosols (i.e., the unattached fraction) is given by:

$$f_a = \frac{\lambda_a}{(\lambda_a + \lambda_s)}$$

$$f_b = \frac{(\lambda_b)}{(\lambda_b + \lambda_s)} (f_a [1 - \alpha_a] + \alpha_a)$$

$$f_c = \frac{(\lambda_c)}{(\lambda_c + \lambda_s)} (f_b [1 - \alpha_b] + \alpha_a),$$

where f_a, f_b and f_c are the unattached fractions for ^{218}Po, ^{214}Pb and ^{214}Bi,respectively the radionuclides that contribute the vast majority of the lung dose. (These isotopes are known historically as Radium A, Radium B and Radium C, respectively.) In these equations, λ_a, λ_b and λ_c are the radiological decay rate constants for RaA, RaB and RaC, respectively, while λ_s is the rate of attachment of atoms to aerosols. The parameters, α_a and α_b, indicate the probability that a decay of an atom of RaA or RaB results in detachment of the atom from any aerosol to which it might be attached prior to decay. Typical assumed values for α_a and α_b are 0.5 to 0.8 and 0.0, respectively (Raabe 1969, Mercer 1976). Values for the unattached fraction of ^{218}Po as a function of aerosol concentration are displayed in Figures II-5 and II-6 and display the agreement between empirical measurements at several sites and the predictive results of Raabe 1969. A recent committee, formed to study the doses delivered from indoor exposure to ^{222}Rn and progeny, employed an unattached fraction of 0.07 for RaA/Rn (Cross 1983), as did the NCRP group studying the same problem (NCRP 1984a) and this value is used in the present report.

The presence of decay chains in air presents a complicated problem if an attempt is made to summarize the potential hazard posed by a single number. This problem arises from the fact that the risk depends not only on the total concentration of the progeny atoms, but also on the relative concentrations of the various progeny. In order to partially circumvent this problem and provide a convenient unit of exposure, Holaday (1957) introduced the concept of the "Working Level". One Working Level (WL) was defined as "any combination of the radon progeny in air which will result in the emission of 1.3×10^5 MeV of potential alpha energy in the complete decay through RaC". Contributions from the decay of ^{222}Rn itself were excluded from the definition due to the insignificance of its delivered dose to the lung (Bale 1951). Cumulative exposure to the radon chain is typically measured in units of Working Level Months (WLM), with one WLM being defined as a

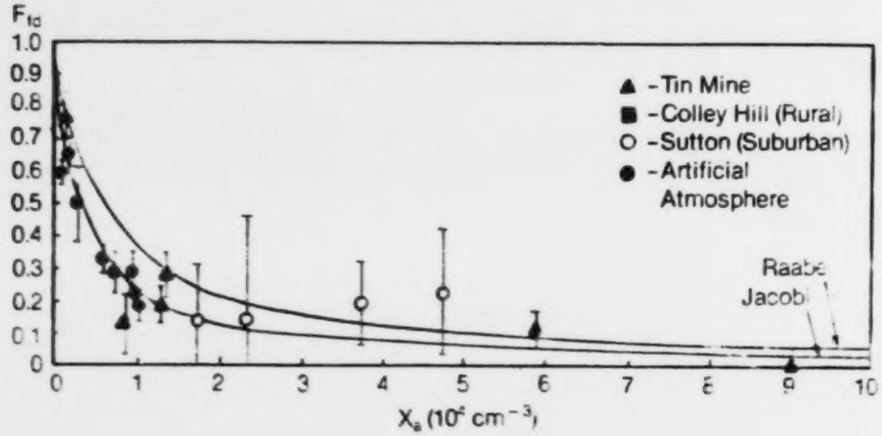
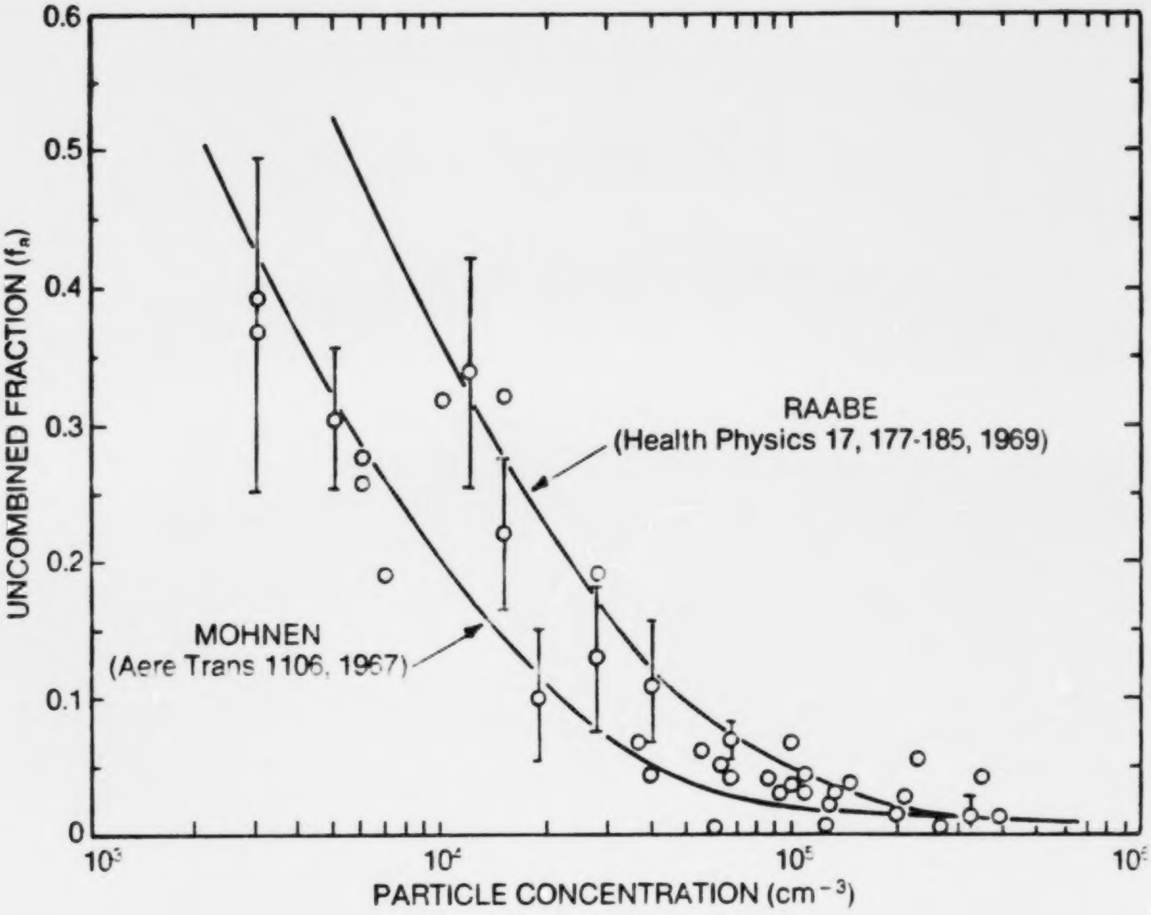


Figure II-5 Variation of the Fraction (F_{fd}) of Unattached ^{218}Po as a Function of the Concentration of Condensation Nuclei (X_a)



Adapted from NCRP 1984a.

Figure II-6 Unattached RaA Fraction versus Aerosol Particle Concentration

cumulative WL for a period of 170 hours (the normal number of hours worked per month).

A concentration of one WL results from 100 pCi/L of ^{222}Rn in equilibrium with its progeny. Using International System units, this corresponds to an equilibrium concentration of all progeny of 3.7 kBq/m^3. Complete equilibrium of all progeny with the ^{222}Rn parent is reached only in instances in which emanation is into a closed volume. When there is ventilation within the volume, the activity concentration of progeny atoms will be less than that for the ^{222}Rn. The growth of the various radon progeny in an enclosed box into which 100 pCi/L of ^{222}Rn is introduced is given in Figure II-7, which assumes no ventilation. Table II-5 reports the build-up of the progeny in room air at various levels of ventilation. It will be noted that the concentrations of the progeny decrease as the ventilation rate increases.

The ratio of the concentration of a given progeny to its ^{222}Rn parent is termed the equilibrium ratio and, from the above, clearly decreases with increasing ventilation. These progeny also tend to deposit on walls and other surfaces, thereby further decreasing the equilibrium ratio for each progeny. As discussed earlier, the progeny exist in both attached (to aerosols) and unattached states in air, with the unattached atoms having greater mobility to any surfaces such as walls and floors. In general, the rate of deposition onto

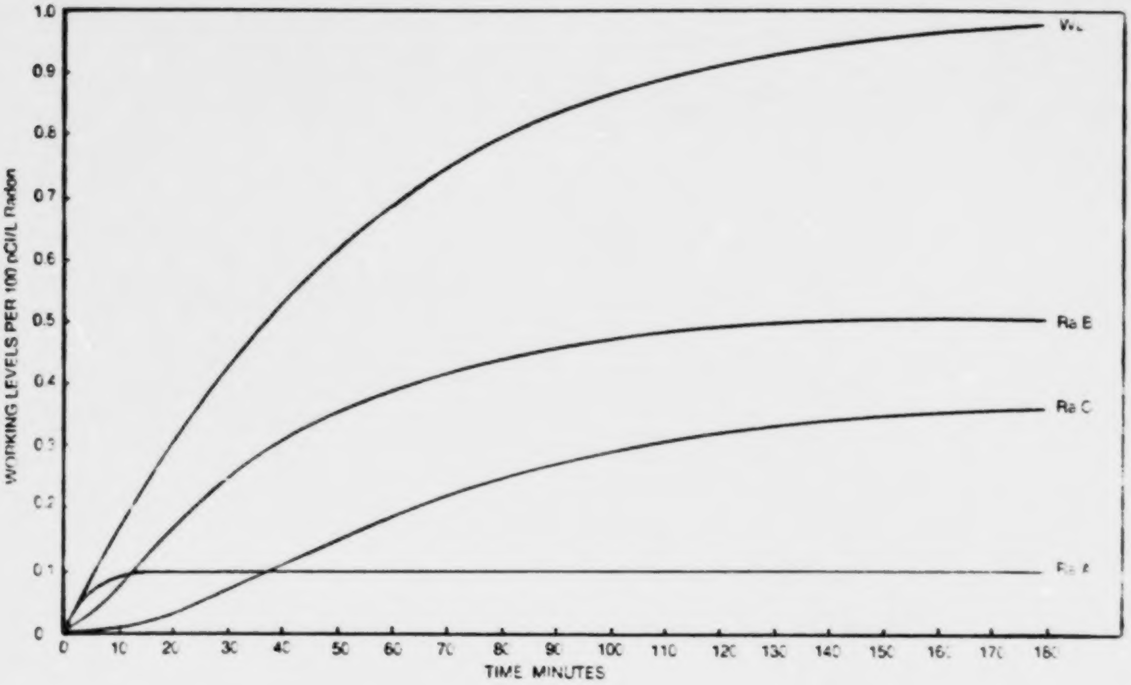


Figure II-7 Growth of WL Concentration of Radon Progeny with Time in an Unventilated Container

surfaces for the progeny is given by

$$\lambda_{d,f} = U_{d,f}\ (A/V)$$

for the free, or unattached, fraction and by

$$\lambda_{d,a} = U_{d,a}\ (A/V)$$

for the attached fraction. Here, $\lambda_{d,f}$ and $\lambda_{d,a}$ are in units of (time $^{-1}$), $U_{d,f}$ and $U_{d,a}$ are the deposition velocities of free and attached atoms, respectively, and A and V are the surface area and volume of the enclosing structure. These values for $U_{d,f}$ and $U_{d,a}$ may be obtained from experimental results (Wells 1967, Montgomery and Corn 1970, Friedlander and Johnstone 1957). Jacobi (1964) set $U_{d,a}$ equal to 10^{-2} cm/second and $U_{d,f}$ equal to 1 cm/second. Results similar to these were utilized by UNSCEAR (1982) to determine values for $\lambda_{d,f}$ and $\lambda_{d,a}$ of 30 hour^{-1} and 0.2 hour^{-1}, respectively, for normal sized rooms.

The concentrations of the individual radon progeny in a volume of air may be related to the definition of the WL through the following equation:

$$\begin{aligned} WL &= 0.00103\ I_a \\ &+ 0.00507\ I_b \\ &+ 0.00373\ I_c \end{aligned}$$

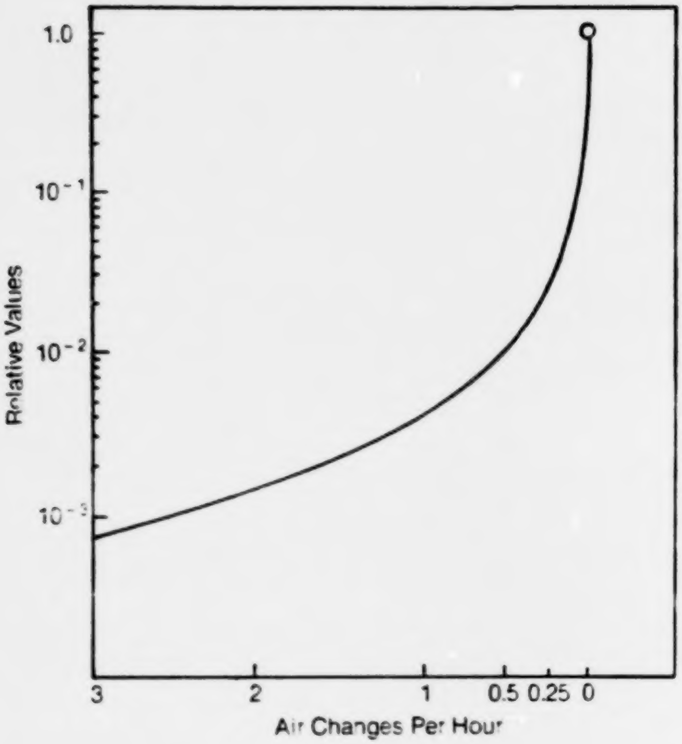
Here, I_a, I_b and I_c are the airborne concentrations of RaA, RaB and RaC, respectively, in units of pCi/L, with no separation of the attached and unattached fractions. From this definition,

Table II-5 Equilibrium Ratios for Radon Progeny and Equilibrium Factor F for Various Ventilation Rates

Ventilation rate, v (h^{-1})	Equilibrium ratio, $x_{a,j}/x_{a,Rn}$			Equilibrium Factor F
	^{218}Po	^{214}Pb	^{214}Bi	
0.0	1.0	1.0	1.0	1.0
0.1	0.993	0.956	0.913	0.928
0.3	0.978	0.820	0.718	0.784
0.5	0.965	0.729	0.590	0.689
0.7	0.951	0.655	0.530	0.628
1.0	0.932	0.566	0.384	0.526
1.5	0.900	0.473	0.277	0.436
2.0	0.872	0.361	0.195	0.356
3.0	0.820	0.279	0.115	0.269
5.0	0.732	0.173	0.051	0.182
10.0	0.577	0.077	0.013	0.103

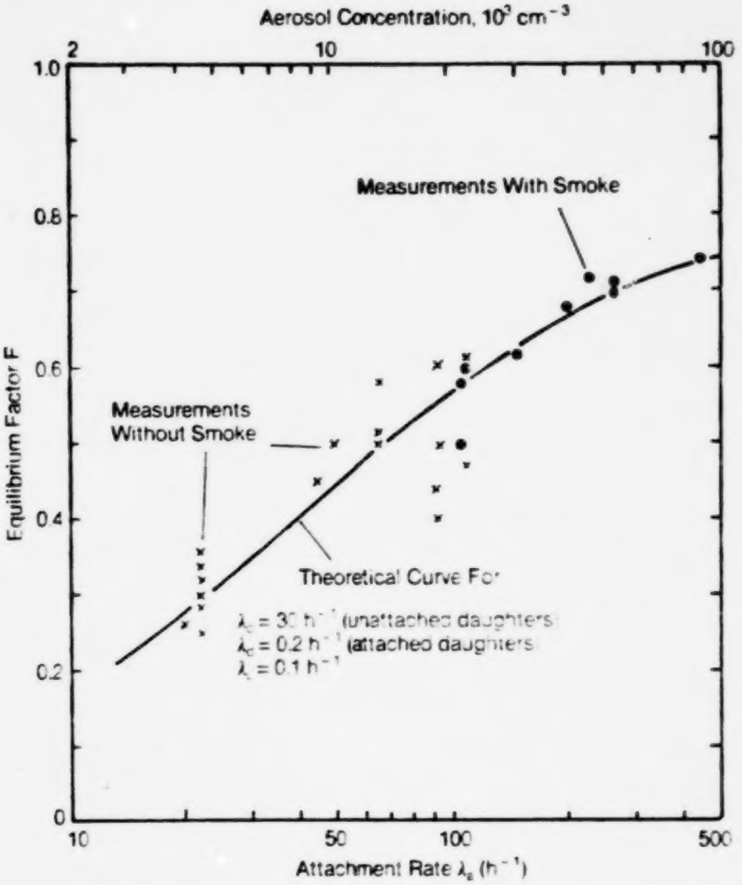
Data from UNSEAR 1982.

it is possible to define an equilibrium factor, F, that is equal numerically to the ratio of the WL concentration of progeny in air for a specified set of conditions over the WL concentration that would hold under conditions of no ventilation or surface deposition (usually called plate out). Calculated relative values of the potential alpha energy ventilation rates are given in Figure II-8, and values of F for a ventilation rate of 0.1 air change per hour and for varying aerosol concentrations are given in Figure II-9.



Adapted from UNSCEAR 1982.

Figure II-8 Potential Alpha Energy in Air versus Ventilation Rate



Adapted from UNSCEAR 1982.

Figure II-9 Equilibrium Factor versus Aerosol Concentration, with an Emanation Rate into the Room of 7.4 Bq m^{-3} hour^{-1}

In summary, then, the radon isotope of primary radiological concern is ^{222}Rn, with dosimetric attention being directed towards the resulting radon progeny, rather than to the inert gas itself. The factors controlling the extent to which these progeny may be found in air are (1) the rate of ^{222}Rn emanation into the location of interest, (2) the local ventilation rate, (3) the concentration of aerosol particles in the air and (4) the degree of equilibrium between ^{222}Rn and its progeny through ^{214}Po. Typical equilibrium ratios (i.e., the relative values of the concentrations of ^{222}Rn: ^{218}Po: ^{214}Bi: ^{214}Po) for dwellings are approximately 1:0.9:0.6:0.35, which arises from a ventilation rate of one air change per hour. Radon exhalation rates into homes from ^{226}Ra in soil and building materials can vary greatly with time for an individual dwelling, subject to such factors as atmospheric pressure and humidity. A recent summary of these conclusions can be found in the report of the National Council on Radiation Protection and Measurements (NCRP 1984a).

Measurements of the equilibrium factor in apartments in the Federal Republic of Germany (Wicke and Porstendorfer 1982) indicate an F value of approximately 0.40, while studies in Austria (Steinhausler et al. 1980), Sweden (Swedjemark 1978, Swedjemark et al. 1979), Canada (Letourneau et al. 1978), Norway (Stranden et al. 1979), Finland (Makelainen 1980) and the United Kingdom (UNSCEAR 1982) indicate F values of 0.62, 0.45, 0.60, 0.38, 0.50 and 0.50, with geometric standard deviations of approximately 1.2 to 1.5. An equilibrium factor, F, of 0.5 is used for calculations in the present report, as this is approximately the average of the reported measurements and is similar to that suggested by the NCRP(1984a). This figure agrees with the most recent results published by Keller and Folkerts (1984) for the air of homes in the Federal Republic of Germany. Their results also show that the equilibrium factor is not a sensitive function of the ventilation conditions within the normal range found in dwellings. For purposes of comparison, a recent committee studying the risks of indoor radon exposures assumed an equilibrium factor of 0.7 (Cross et al. 1985), resulting in only a 5% change in risk estimates from those obtained with an equilibrium factor of 0.55. When the equilibrium factor dropped below 0.4, however, they found a rapid change in risk estimates as a function of equilibrium factor.

In all calculations in the above committee report (Cross et al. 1985, NCRP 1984a) and in the present report, the unattached fraction of RaA in indoor home air is taken to be 7%. This contrasts with the typical value of 4% found in mines, where the density of aerosol particles is higher. Attached radon progeny deposit in the lung passages with a probability of only a few percent, while the unattached progeny deposit with nearly 100% probability due to their much greater diffusion coefficient. Fortunately, the unattached fraction in dwellings and other buildings appears to be reasonably constant (Cross et al. 1985).

B. ANALYSIS OF RADON IN DRINKING WATER

The fact that radon is an inert gas facilitates its transfer from a sample of water to any detection device used to measure the radon. As a result, several methods (discussed below) have been developed for measuring radon content in water through de-emanation of the gas into scintillation flasks or Lucas cells (Lucas 1957). Both inorganic and organic scintillators have found application in this measurement of radon. These methods rely on the fourfold increased solubility of radon in air over that inherent in water (Schulze 1920).

Typically, the water sample is connected to an air pump, and air is bubbled through the sample holder (Prichard 1978) at a controlled rate. The partially dissolved radon is carried with the air and enters a Lucas cell for counting. The Lucas scintillation cell (Lucas 1957) is an evacuated chamber (typically 100 mL in volume) with interior surfaces coated with a ZnS:Ag scintillator. One inner surface, consisting of a quartz window, is uncoated, allowing the scintillations produced by alpha emissions to leave the cell. The dimensions of the flask are such that almost all alpha particles given off by radon and progeny will reach the phosphor if the air is dry and at Standard temperature and pressure (STP). The resulting light pulses are collected by a photomultiplier tube and counted by a scaler.

Calibration of the system is performed using a radon standard, which typically consists of a source of radium in water producing a constant (and known) activity of radon per unit time. Care is taken to ensure that water samples are not exposed to air prior to measurement, because significant fractions of the dissolved radon can escape prior to counting. Discriminating against low-amplitude background pulses from the photomultiplier tube allows only the alpha decays from radon and progeny (usually allowed to grow into essentially complete equilibrium) to be counted. As a result, background levels can be lowered to 0.1 counts/minute. Overall system efficiency is 75% to 80% for the alpha decays (Prichard 1978), with the loss being due primarily to incomplete energy transfer from the alpha particles to the scintillator and to rejection by the discriminator. In addition, since small leaks are difficult to avoid, some radon may be lost during the period of time allowed for ingrowth of progeny prior to counting.

Liquid scintillation counters have also found wide use in measuring radon in water. This approach relies upon the relatively high solubility of radon in certain hydrocarbons that can be used as solvents in a liquid scintillation vial (Schulze 1920, Haseltine 1970). The radon can be measured either in its original concentration, by direct injection of the water sample into the liquid scintillation fluid (Prichard and Gesell 1977, Horton 1983), or by pre-concentration, by extraction of the radon from large volumes into smaller scintillation volumes (Noguchi 1964, Homma and Murakami 1977). The former method provides for the easiest and most rapid determination in large numbers of samples, with clear capability to measure radon-water concentrations as low as 10.0 pCi/L (Gesell 1977). For much lower concentrations, high volume extraction offers greater sensitivity, although this increased sensitivity should not be needed to meet regulatory limits.

The U.S. Environmental Protection Agency (USEPA 1978) has made extensive use of the direct liquid scintillation method in its sampling program. In this procedure, a 10-mL aliquot of the water sample is drawn into a syringe and transferred to the liquid scintillation vial containing the scintillator (mineral oil-based). Problems arise in ensuring that the radon does not leave the sample in the syringe, thus resulting in an unrealistically low estimate of the radon concentration in the original sample. Kahn et al. (1983) recommends that the vial sample be obtained under conditions of positive pressure to retard the de-emanation of the radon into the air.

A final method employed in estimating radon content in water relies upon detection of the gamma radiation emitted by the radon progeny (Lucas 1964). While this method is very simple to employ, the gamma radiation detection efficiencies for either NaI or GeLi detectors are much lower than those obtained by alpha counting, and the background count rates for the system can be comparatively large. As a result, signal to noise ratios can be large for small samples and short counting times. This implies that either large volumes of water or long counting times must be employed. As a result, this method is not commonly found in programs designed to sample many bodies of water.

In summary, liquid scintillation counting of water samples appears to afford the greatest ease of measurement, as well as offering acceptable accuracy at environmental levels as low as 10 pCi/L of ^{222}Rn. The ease of preparation arises from the much greater solubility of radon in organic solvents (such as toluene, xylene or psuedocunone) than in water, which is highly polar. This increased solubility is fairly constant, changing by only 4% over a temperature range from 20°C to 30°C. Commercially available organic scintillators serve as excellent scintillation media for this application, with the light pulses from alpha particles being detected with nearly 100% efficiency. One slight problem is that the electron pulses from background radiation are of a height similar to those from the alphas, so discrimination against background by electronic discriminators is troublesome. This problem can be partially remedied, however, by shielding the liquid scintillation detector or by pulse-shape analysis. In any event, acceptable sensitivity to as low as 10 pCi/L should be obtainable by generally accepted health physics procedures. In fact, the ubiquitous use of liquid scintillation by industry and laboratories should prove to be a powerful asset.

III. PHARMACOKINETICS

When radon and its progeny enter a dwelling through water supplies, three primary modes of exposure occur. These are (1) exposure of the lung tissue to the radioactive decays of radon progeny deposited in the lung; (2) exposure of the other body organs to the radioactive decays resulting from ingested radon that subsequently translocates to those organs and (3) external exposure of the body to radiations emitted by radon and progeny located in the air or on exposed surfaces of the dwelling. These three exposure modes will be discussed in the order listed, as this is also the order of decreasing radiological significance.

A. Dosimetry of the Lung

The volume of air which passes into the lungs enters through either the nasal passage or the mouth. These two passages join directly beneath the tonsils, in a zone termed the oropharynx. From this point, the air passes into the trachea and then through a series of bifurcating passageways whose diameters become progressively smaller. The lung proper is typically pictured as consisting of three compartments (Horsfield 1978): a conducting zone, in which gas is transported but not exchanged, a transitional zone, in which there is both transport and exchange, and a respiratory zone, in which only gas exchange occurs. The terminology used to label these regions and the various subdivisions employed vary between models and between authors. Therefore, as much general information as possible (i.e., information which is not dependent on the choice of model) will be provided before turning to specific models.

All models begin with a trachea, which in the adult is a thin-walled, flexible tube, approximately 12 cm in length and with a diameter of 2 to 2.5 cm (Bloom and Fawcett 1968). Its structure is supported by 16 to 20 C-shaped hyaline cartilages which encircle it on its ventral and lateral faces. Flexibility is attained due to the fibroelastic tissue which bridges the incomplete cartilagenous rings. These rings, in turn, are surrounded by a dense matrix of connective tissue.

The inner surface of the trachea, as in most of the bronchial passages, consists of pseudostratified columnar epithelium with a coating of cilia. This layer rests on a thick basement lamina, which acts as a terminus for the basal cell layer. These basal cells are the source of new epithelial cells; they divide when a depletion of columnar cells occurs, then migrate and mature. Goblet or mucus-secreting cells are scattered throughout the columnar cells and are virtually the only examples of unicellular glands found in mammals. Their role is the secretion of mucin, a polysaccharide which combines with water to form a lubricating solution called mucus.

The trachea divides into two main bronchi, each of which enters either the right or left side of the lung at the hilar region. These main bronchi divide into two smaller bronchi on the left side and into three on the right, yielding two and three lobes, respectively. These divisions then give rise to smaller bronchi, which then divide into several orders of bronchioles. These subdivisions continue until one encounters the terminal bronchioles, from which branch the respiratory bronchioles, alveolar ducts, alveolar sacs and, finally, the alveoli, in which carbon dioxide and oxygen are exchanged.

At the level of the bronchi, the cartilagenous rings of the trachia disappear and are replaced by a series of cartilage plates which completely encircle the bronchi. These plates serve to retain the cylindrical shape of the tubes during respiration. They become irregularly distributed as one moves toward the more distal parts of the lung and are replaced by smooth muscle. When the diameter of the bronchiole falls below 7 mm, the cartilage completely disappears.

The cellular structure of the lung passageways remains roughly invariant to the level of the terminal bronchioles. As mentioned previously, this structure consists of basement membrane, immediately above which lies the basal layer, followed by several layers of pseudostratified columnar epithelium. The uppermost layer of cells contains both goblet cells and ciliated epithelium. As demonstrated by Sadé et al. (1970), the goblet and ciliated cells are coupled through the mucus, since a minimum amount of mucus is needed for ciliary activity. Movement of the mucus, whose role is, in part, to provide protection of the epithelial surface by trapping and removing irritants, is provided by the cilia of the uppermost layer of cells. Mucus flow, which has been reviewed by a number of authors, (Van As and Webster 1974, Blake 1973, Laurenzi 1973) is propelled by the beating of the cilia, which consist of the typical 9 + 2 fibril arrangement that causes bending in the cilium by interaction between adjacent fibrils (Sleigh 1962) which is presumably activated by the breaking of ATP near the basal bodies (see Figure III-1).

The functional unit of the lung begins with the respiratory bronchiole and continues to the alveoli. Preceding sections of the lung serve to filter the air, humidify it and provide a division of the air onto a large surface area. Estimates of the surface area of the alveoli indicate roughly 40 to 100 m^2 (Horsfield 1978) of surface interface between the air sacs and the pulmonary circulatory system. This interface is accomplished through a division into upwards of 300×10^6 alveoli (Angus and Turbeck 1972).

Little attention will be focused on the distal section of the lung, for reasons which will become clear later. A brief review is in order, however, simply to complete the description of the lung. The alveolar surface is covered by two primary

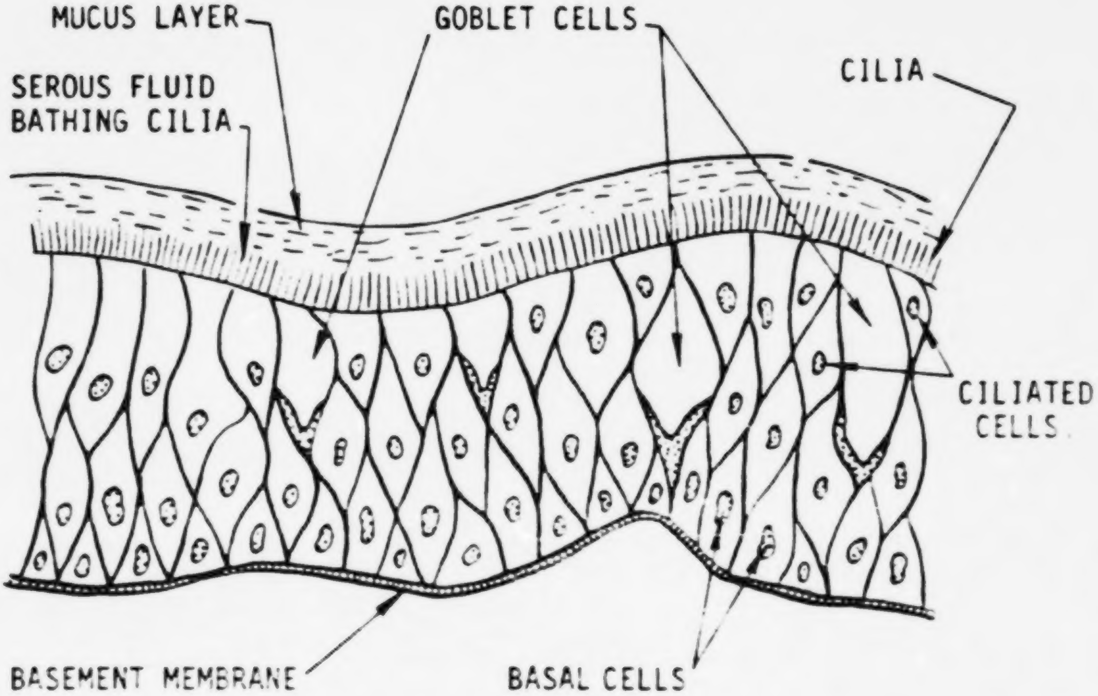

MUCUS LAYER
GOBLET CELLS
SERUS FLUID
BATHING CILIA
CILIA
CILIATED CELLS
BASEMENT MEMBRANE
BASAL CELLS

Figure III-1 A Portion of the Bronchial Epithelium Shown in Cross Section

types of cells joined by tight junctions (Kuhn 1976, Weibel 1973) which prevent passage of material between the cell walls. The first is the highly specialized cell (Weibel 1976) known as the squamous or Type I epithelial cell. It possesses the ability to extend thin sheets of cytoplasm radially, thereby creating a large surface area. Of course, this large surface area, estimated to be approximately 50 times that of the Type II cell per cellular unit (Weibel 1976), makes it more susceptible to damage. The Type II alveolar cell, or granular pneumonocyte, acts as the source of the surface-active phospholipids in the alveolar lining (Jeffery and Reid 1978). These cells also divide to form Type I cells when a stimulus causes the Type I population to decline.

Alveolar cells are in close alignment with the endothelial cells of the pulmonary circulatory system. These groups of cells are separated by only a thin layer of interstitial cells, which in some cases may even disappear. This proximity greatly facilitates the transfer of carbon dioxide and oxygen across the membranes. For a good review of these processes, the reader is referred to Comroe (1965) and Kuhn (1978). The stem cells for the alveoli and the upper respiratory tract have been shown to be the Type II alveolar cells and basal cells, respectively. In the respiratory bronchioles, which reside in the transitional region, replacement of the ciliated cells is accomplished through division and transformation of the mucus-secreting cells. Therefore, if any credence is to be given to the Law of

Bergonie and Tribondeau in this context (Cassarett 1968), the most sensitive lung cells should be the basal cells in the upper lung (although the K cells also have been implicated recently, NCRP 1984a), the goblet cells in the transitional region and the Type II cells in the alveoli.

It should be clear that the lung is a complex organ with a large potential for variability due to uncertainty in the appropriate conceptual scheme for dealing with the multiple branching. For calculational purposes, this variability (as well as variation in other factors) has led to the proposal of a number of lung models, each of which may differ in the number of regions employed, the number of tubes in each region, the angle between branches and the length of each region. In addition, a choice is usually made between dichotomous branching in a symmetric tree and non-symmetric branching. As mentioned earlier, the difference in lobe number between the right and left lung (three and two, respectively) suggests that the non-dichotomous scheme may be more correct by this criterion.

Six lung models have been dominant in historical calculations, primarily due to their relative accuracy and/or ease of use. These models were developed by Weibel (1963), Davies (1960), Landahl (1950), Findeisen (1935), Horsfield (1971) and Yeh and Schum (1980), with the Weibel, Findeisen and Landahl models receiving the widest use until recently. The Landahl model is actually a modified version of the Findeisen model, and its parameters are given in Table III-1. The Weibel model is believed to be more anatomically correct, and the parameters associated with it are shown in the same table. The most recent studies of radon progeny deposition employ either the Weibel (1963) or Yeh and Schum (1980) morphologies.

Since radon is an inert gas, its entry into the lung does not result in any deposition onto the lung walls unless the radon decays to one of the progeny while in the lung. As a result, the dose to the lung under environmental conditions is small for the radon itself (compared to the dose from inhaled progeny). In addition, the absorption of radon in body tissues leads to a similarly small dose. The progeny, however, exist in air either attached to aerosol particles or as free charged ions. As a result, they attach to any lung wall surfaces which might intersect their paths. Three main processes contribute to the deposition of these radon progeny, with the relative contributions being a function of the size distribution of any inhaled aerosols and of the unattached fraction. These processes are impaction, sedimentation and diffusion (or Brownian motion), with some minor contribution from depostion of charged particles by the image force resulting from rearragement of charge on the surface of lung passages. (Yu and Changra 1978). The latter mechanism is not usually included in calculational models.

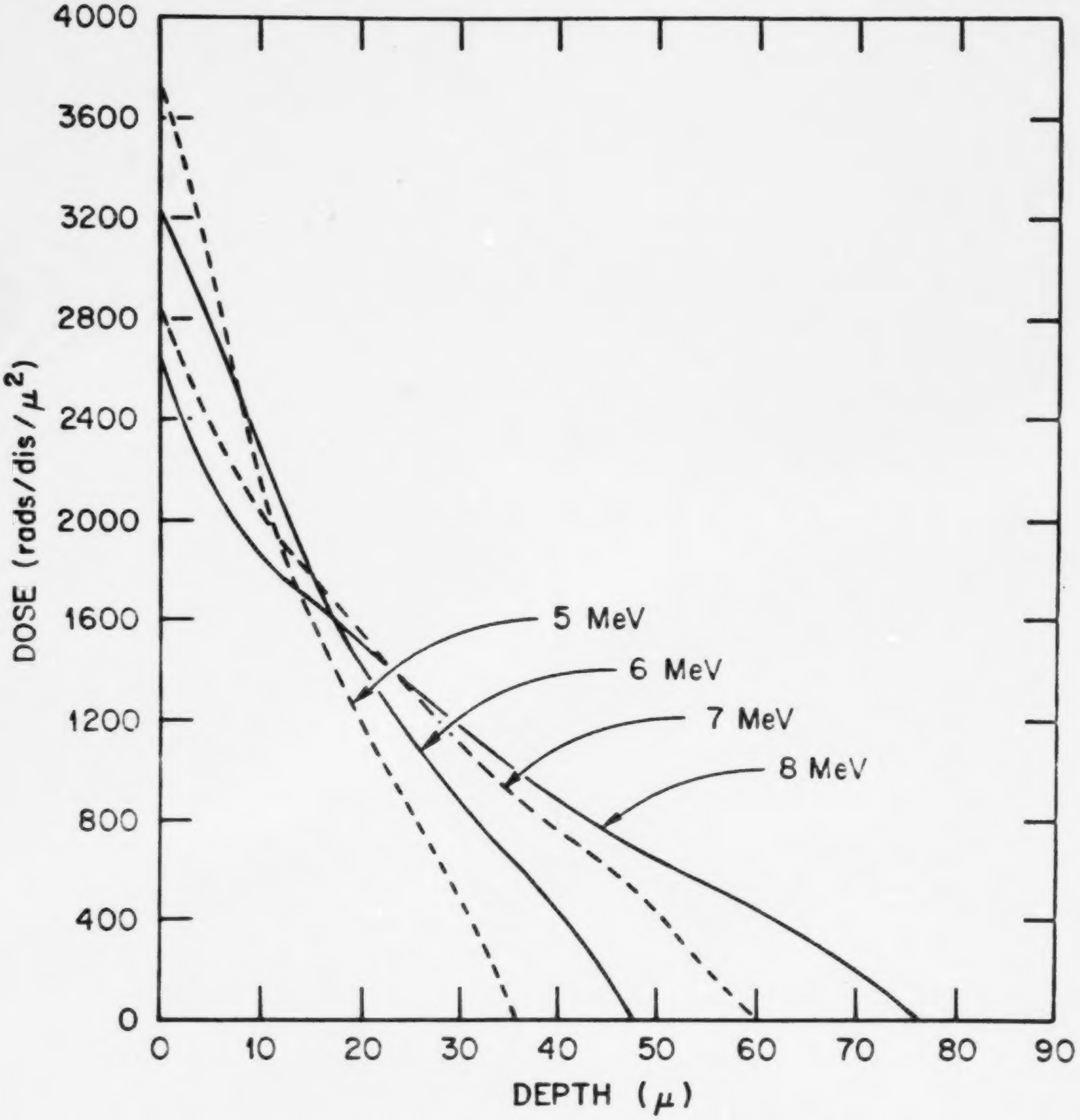
Table III-1 Model Parameters for the Findeisen-Landahl[a] and Weibel[b] Models

Region	Number	Diameter (cm)	Length (cm)
	Findeisen-Landahl		
Mouth	1	2	7
Pharynx	1	3	3
Trachea	1	1.6	12
Primary Bron.	2	1	6
Secondary Bron.	12	0.4	3
Tertiary Bron.	100	0.2	1.5
Quartenary Bron.	770	0.15	0.5
Terminal Bron.	5.4×10^4	0.06	0.3
	Weibel		
0	1	1.8	12.0
1	2	1.22	4.76
2	4	0.83	1.90
3	8	0.56	0.76
4	16	0.45	1.27
5	32	0.35	1.07
6	64	0.28	0.90
7	128	0.23	0.76
8	256	0.186	0.64
9	512	0.154	0.54
10	1,024	0.13	0.46
11	2,048	0.109	0.39
12	4,096	0.095	0.33
13	8,192	0.082	0.27
14	16,384	0.074	0.23
15	32,768	0.066	0.20
16	65,536	0.06	0.165

[a] Findeisen (1935).
[b] Weibel (1963).

Mathematical models for calculating deposition of radon progeny can differ in the choice of any of the three equations above, in the anatomy of the lung or in breathing patterns. For a complete review of many of these considerations, the reader is referred to any of several publications (Walsh 1970, Crawford 1982, 1980). Once the radon progeny have been deposited within the bronchial passages, they are partially removed by the mucociliary blanket, which carries them toward the trachea and, ultimately, to the G.I. tract. Decay of these progeny occurs during this transport. Calculations of lung dose from inhaled radon progeny can also employ differing assumptions concerning the rate at which these progeny are carried by the mucus through any given region of the lung and the distribution of the progeny within the thickness of the mucus layer.

When the modeler has determined the spatial pattern of radon progeny decays on the surface of the lung passages, the doses to underlying tissues may be computed (see Figure III-2) from a knowledge of the stopping power curves (Walsh 1970) for the appropriate alpha emissions (which deliver the large majority of the dose). Problems arise at this step in specifying the particular lung cells that ought to be considered at risk. Current thinking in radiobiology (NCRP 1984a) places these critical cells in the bronchial epithelium of the lung, with particular emphasis on the basal cells and, possibly, the K cells. There is still some controversy over the precise



Adapted from Crawford 1982.

Figure III-2 Depth-Dose Curves (rads per alpha disintegration per square micron of lung surface) in a Tracheobronchial Passage of Radius R = 1 cm and for Various Alpha Energies (5-8 MeV)

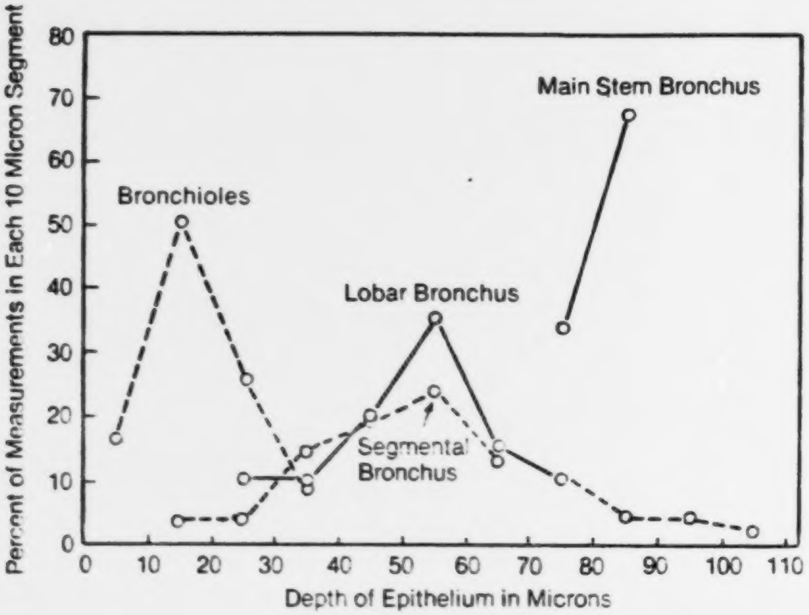
location of these cells in most of the generations of the lung, and different researchers have employed different assumptions. Again, reference to Figure III-2 indicates that computation of dose depends strongly on the chosen location of the critical cells.

In the past five years, six primary studies (presented below) have been performed relating radon daughter exposures and doses to critical lung cells. The results of each are presented in units of dose per WLM. Given the large number of possible assumptions that go into the calculational models, these six results are in substantial agreement for calculations of both mining and environmental (home) exposures. In general, these studies have calculated that the bronchial epithelium cumulative exposure-to-dose conversion factor is 0.5 to 0.8 rad/WLM for the uranium miners.

Environmental exposures, however, are characterized by different particle sizes, fraction of unattached RaA (7% environmental, 4% in mines) and breathing rates than were true for the uranium miners. These changes have been incorporated into the six model calculations discussed here.

Harley and Pasternack (1981), in calculations that form the basis for the recent NCRP report (NCRP 1984a), calculated the exposure-todose conversion factor for environmental exposures, assuming an equilibrium factor, F, of 0.7. They obtained values of 0.71 rad/WLM for the adult male and 0.64 rad/WLM for the adult female. These authors calculated doses to the basal cells located at an assumed constant depth of 22 um below the epithelial surface. This latter assumption contrasts with the other studies, which employ the basal-cell-depth distributions of Gastineau et al. (1972). In this latter case, the epithelium thickness and, hence, the location of the basal cells below the bronchial surfaces decrease as the size of the passages decreases (see Figure III-3).

Hofmann (1982a,b) used the Gastineau data, along with slightly different assumptions of unattached fractions, breathing rates, anatomical structure, deposition equations and mucociliary transport rates. His estimate of the radon progeny exposure-to-dose conversion factor is 0.6 rad/WLM to the basal cells of the bronchial epithelium. Other recently published results are those of Jacobi and Eisfeld (1980) who found a conversion factor of between 0.4 and 0.6 rad/WLM; James et al. (1980, 1981) calculated a conversion factor of 0.3 to 1.0 rad/WLM; and the International Commission on Radiological Protection (ICRP 1981) published a conversion factor of 0.3 to 0.8 rad/WLM. In a review of these calculations, a recent committee studying the health effects of inhaled radon (Cross et al. 1985) concluded that a "reasonable, mean, environmental conversion factor [of] 0.7 rad/WLM for bronchial epithelium" would be adopted for their report. Given the good agreement of the previous results, and despite modeling complexities, this conclusion appears to be strong.



Adapted from Gastineau 1972.

Figure III-3 Basal Cell Depth Within Various Generations of the Tracheobronchial Region

It should be borne in mind that these calculations focus entirely on the dose to the bronchial epithelium. This feature arises from two considerations. First, the carcinomas in the uranium miners appear to be arise at some location in the bronchial epithelium, suggesting that this region contains the critical cells of radiobiological interest. In addition, the dose delivered to the alveolar region from an exposure to the radon progeny is significantly lower than that in the bronchial region. As a result, the risk to the lung from inhaled radon progeny should be dominated by the risk to the bronchial epithelium.

The above conversion factors apply only to adult occupational exposures to radon progeny. Radiological concern, however, should include exposures to younger age groups, particularly in light of recent calculations that indicated the potential for significantly increased risk at very young ages (relative to the adult risk). Cumulative exposure-to-dose conversion factors for children differ from those for adults due to age-dependent changes in lung anatomy and breathing patterns (Crawford 1982, Hofmann 1982a). For example, Figure III-4, taken from a paper by Crawford (1982), indicates the effect of age on deposition of variously sized aerosol particles in the human lung. Figure III-5, taken from the same paper, displays the effects of age on the number of disintegrations that occur per gram of lung tissue. Clearly, there are noticeable differences between values for adults and

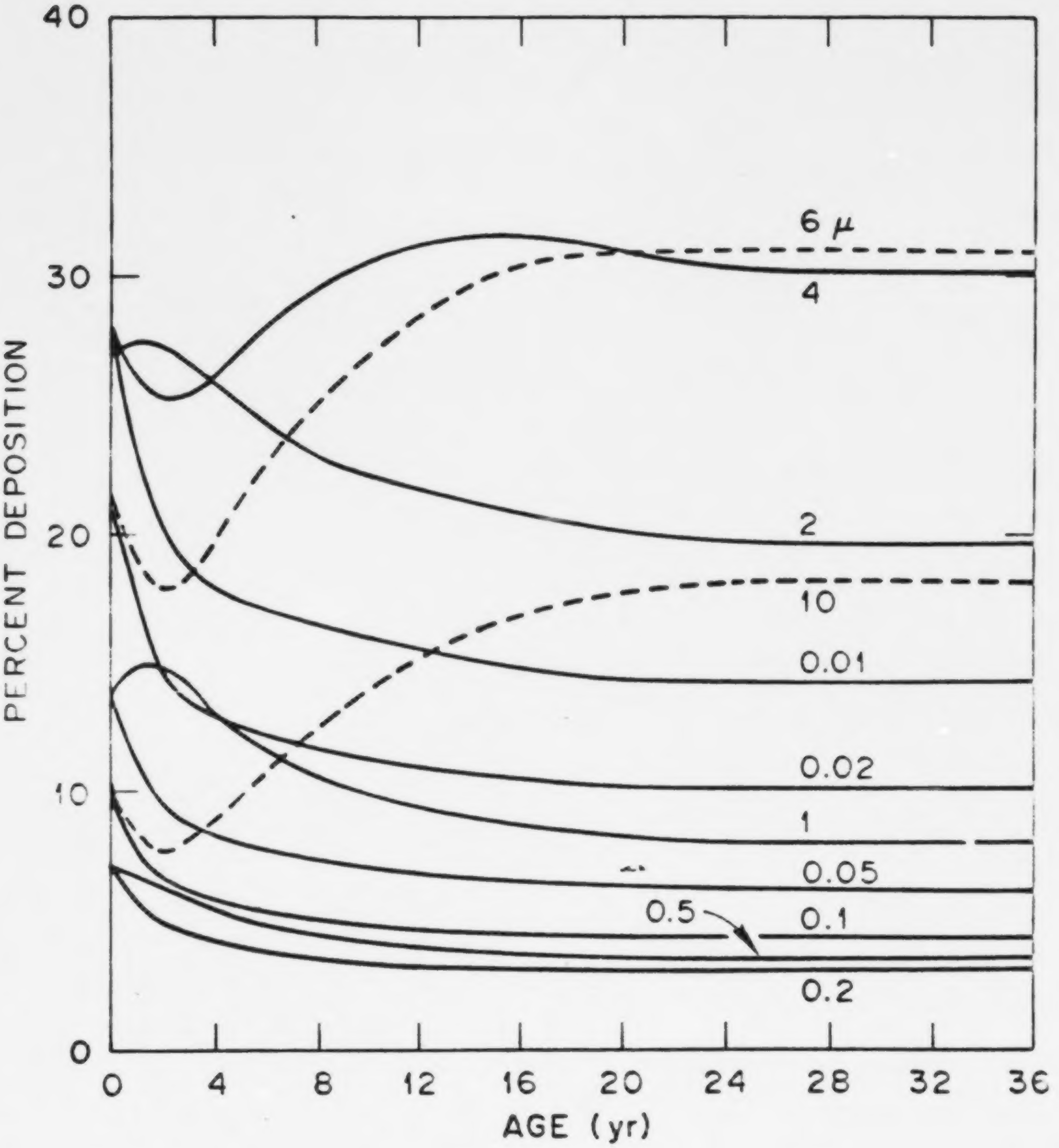


Figure III-4 Tracheobronchial Deposition Versus Age for Variously Sized Aerosols

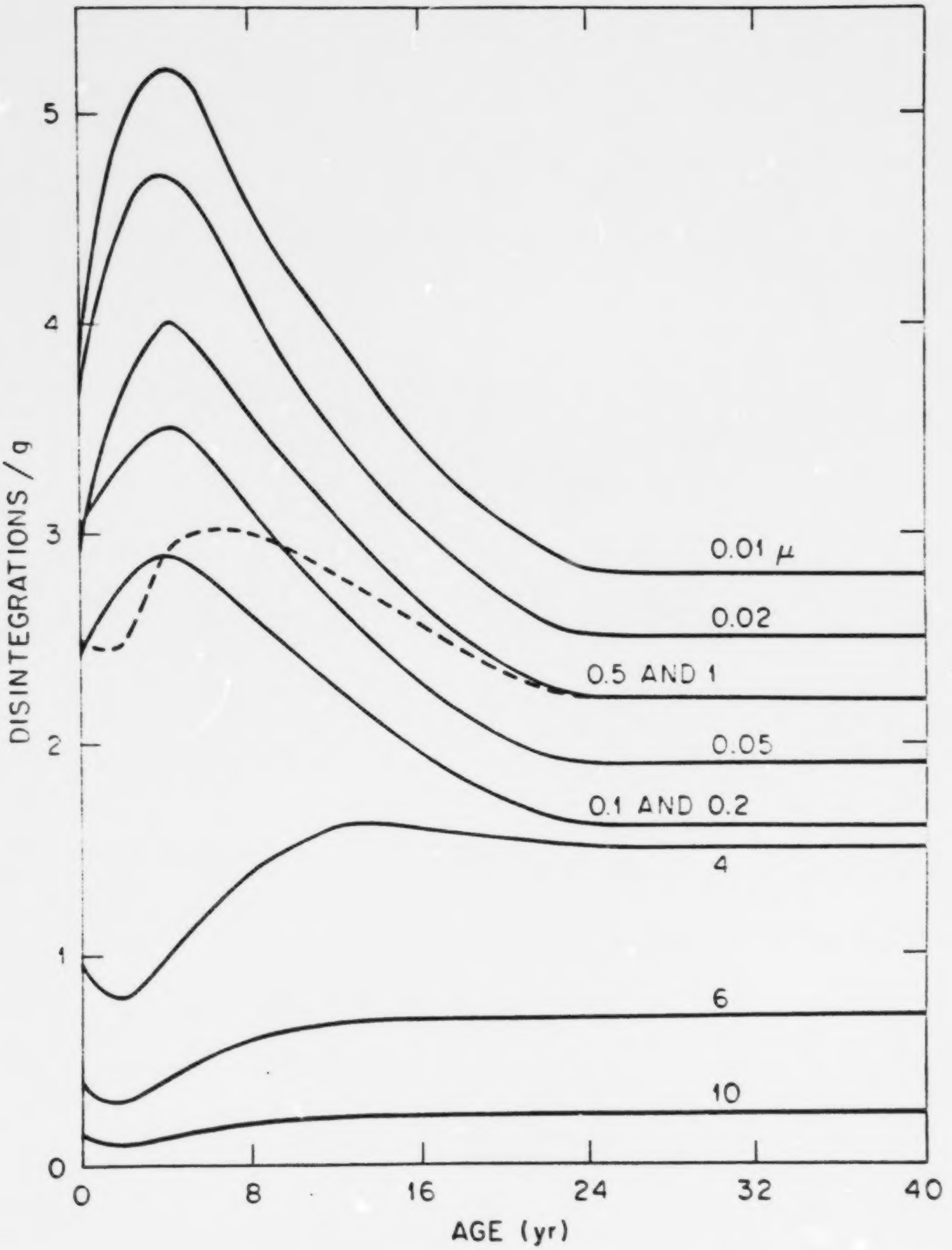


Figure III-5 Relative Measure of Tracheobronchial Dose for Various Ages and Aerosol Sizes

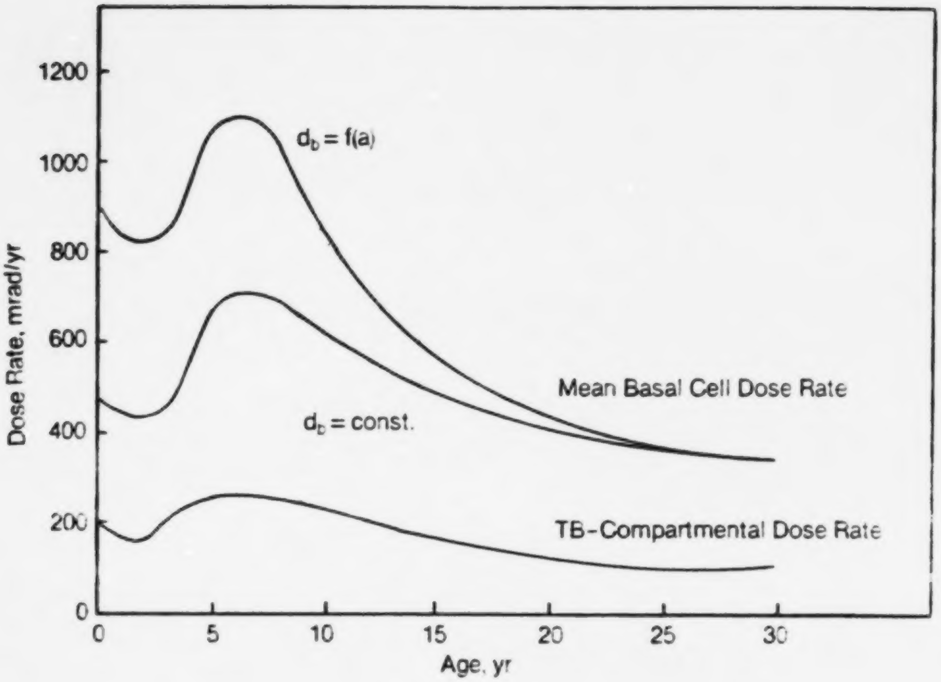
children, particularly in the size range of interest here (AMAD 0.05 to 0.1 um).

Harley and Pasternack (1981) modeled these considerations of age and reported conversion factor values of 1.2 rad/WLM for the 10-year-old child and 0.64 rad/WLM for the 1-year-old child. Hofmann (1982a) has demonstrated that the basal cell doses in the general bronchi for children in the first ten years of life are higher than those in adults by a factor of two to three. The study by Hofmann (1982a) of the assessment of age dependence is displayed in Figure III-6.

B. Dose from Radon

The data and models available for assessing the dose delivered from ingested radon are much less developed than those for inhalation. This is partially due to the historical lack of concern over ingested radon. In the following discussion, it should be borne in mind that this document focusses only on the radon itself which is ingested, and not on radon progeny which might be ingested along with radon.

A few studies have been performed to determine the extent to which ingested radon alone may deliver doses to internal organs. Suomela and Kahlos (1972) used human volunteers to measure empirically the dose equivalent delivered following ingestion of radon. Their results, obtained through whole body counting of the volunteers, indicate a dose to the stomach wall of 24 mrad/ microcurie of ^{222}Rn when the stomach is



Adapted from Hofmann 1982a.

Figure III-6 Mean Basal Cell Dose Rates as a Function of Age at Exposure. Top curve assumes basal depth changes with age. Middle curve assumes no change with age.

full and 38 mrad/ microcurie of ^{222}Rn when the stomach is empty. These values would be converted to dose equivalents of 240 mrem/microcurie of ^{222}Rn for the full stomach and 380 mrem/microcurie of ^{222}Rn for the empty stomach if a quality factor of 10 were applied for alphas. In keeping with more recent recommendations of ICRP (1977), a quality factor of 20 would raise these values to 480 mrem/microcurie of ^{222}Rn (full stomach) and 760 mrem/ microcurie of ^{222}Rn (empty stomach). Suomela and Kahlos (1972) concluded that the risk to the stomach wall dominates over the risk to other organs following ingestion of radon.

Similar conclusions were drawn by Hursh et al. (1965), who calculated the stomach dose from ingested ^{222}Rn. Of particular importance here is the calculation showing that the dose contribution from the short-lived radon progeny (produced following ingestion) is small compared to that of the radon itself. If a quality factor of 20 is applied to their estimates, the conversion factors for ingestion doses to the stomach wall are 412 mrem/ microcurie of ^{222}Rn and 440 mrem/microcurie of ^{222}Rn for full and empty stomachs, respectively. The authors also reported an annual stomach dose equivalent of 2.4×10^{-5} rem/pCi/L of ^{222}Rn if a daily consumption of 1,200 mL of water is assumed.

Anderson and Nilsson (1964) have studied the doses received following ingestion of water in Sweden. Two subjects ingested tap water containing 69,000 pCi/L of ^{222}Rn and were then monitored by whole body measurements. At equilibrium in the body, a dose equivalent rate of 0.1 rem/week averaged over the whole body mass was determined, although the dose equivalent rate to the stomach probably is significantly higher.

Von Dobeln and Lindell (1964) also determined stomach and whole body (these historical studies employ the term whole body to include the entire mass of soft tissue) doses following radon ingestion and concluded that doses to the stomach dominate. Their results indicate a conversion factor of 4 mrem/microcurie of ^{222}Rn for the whole body and 400 mrem/microcurie of ^{222}Rn for the stomach. Their value for the whole body is lower than that obtained by Anderson and Nilsson (1964), who calculated a whole body dose equivalent of 14 mrem/microcurie of ^{222}Rn ingested.

In the most recent review and development of the dosimetry and metabolism of ingested radon, Dundulis et al. (1984) have determined that the experimental results of Suomela and Kahlos (1972), Hursh (1965) and Andersson (1964) indicate a biological half-life for ingested radon of between 30 and 70 minutes in the adult human body, depending on whether the stomach is full or empty. They used these results to modify the ICRP G.I. tract model and then calculated the dose equivalent delivered to body tissues following ingestion of radon-laden water. Their focus was on the regions of the G.I. tract, and they determined that the relative dose equivalents to the four segments are 0.16 to the stomach, 0.096 to the small intestine, 0.226 to the upper large intestine and 0.517 to the lower large intestine. They point out, however, that their model does not include diffusion from the large intestine, resulting in a probable over-estimate of doses to this region.

Assuming a daily consumption rate of 300 mL of unheated water, the calculations of Dundulis et al. yield an estimated effective annual dose equivalent (see ICRP 26 publication, ICRP 1977) to the G.I. tract of 7.1×10^{-3} mrem/year/pCi/L of ^{222}Rn in ingested water. To compare this value with previous results, it may be shown that the above result yields an estimate of stomach dose equivalent of approximately 65 mrem/ uCi of ^{222}Rn ingested. This is lower than the previously cited results for the stomach by a factor of between 5 and 15. The major discrepancies appear to arise due to the decision of the previous investigators to employ a longer retention time for ^{222}Rn in the stomach or other parts of the G.I. tract, presumably in an attempt to be conservative. As an aside, Sullivan and Nelson (1982) essentially concur with the calculations of Dundulis et al. (1984), since they state that ingestion doses to the G.I. tract as reported in the earlier studies may be over-estimates by as much as two orders of magnitude.

The above dose estimates from ingested radon may be summarized through a discussion of a general model of the kinetics of radon transport throughout the human body following ingestion. As mentioned above, several authors have reported retention functions for ingested radon in the whole body (i.e., undifferentiated as to location in particular organs). Common to these reports is the finding that ingested radon is eliminated from the body with a half-time of 30 minutes to one hour. In addition, measurements of exhaled air indicate that the radon in lung air reaches a maximum at 5 to 15 minutes, suggesting rapid equilibration within the body for all components except fat, which accumulates radon to a higher degree than other tissues. This equilibration proceeds by absorption of the radon from the G.I. tract into the bloodstream, followed by movement into the liver and lung tissue and, ultimately, into the other body tissues.

The transfer of the radon from the stomach to the body organs depends markedly on the contents of the stomach (Von Dobeln and Lindell 1964). The effects of meals on radon absorption were demonstrated by Meyer (1937) and Hursh et al. (1965), with several maxima occurring in the exhaled radon due to the increased intestinal absorption as food enters the various segments of the G.I. tract. In general, radon removal rates from the stomach decrease as the stomach contents increase. In addition, increased fat or oil content in the stomach tends to decrease the rate of removal of radon to the blood (Von Dobeln and Lindell 1964).

Von Dobeln and Lindell (1964) also reviewed evidence relating radon solubility in fat and water. They concluded that radon solubility in fat is increased over that in water by at least an order of magnitude. This suggests that the equilibrium radon content in body fat should be significantly higher than that in other tissues of the human body. This theoretical suggestion has been borne out by direct experimental observation (Nussbaum and Hursh 1957, Von Dobeln and Lindell 1964, Hursh et al. 1965), in which the distribution coefficient in body fat was determined to be an order of magnitude above that of other organs. As a result, whole body retention curves for ingested radon become dominated by the "fat" compartment after 100 to 200 minutes, with more than 90% of the body burden being found in the fat (Hursh et al. 1965) at this time.

Whole body retention functions for ingested radon have been determined and reported by several authors. Hursh et al. (1965) have given this function as

$$R(t) = 0.612e^{-0.062t} + 0.340e^{-0.0371t} + 0.048e^{-0.00394t}$$

for the case of an "empty" stomach and

$$R(t) = 0.392e^{-0.0578t} + 0.510e^{-0.0116t} + 0.098e^{-0.00231t}$$

for the case of a "full" stomach, where t is in minutes. Harley et al. (1951) calculated a whole body retention function of

$$R(t) = 0.075e^{-109t} + 0.092e^{-9.3t} + 0.184e^{-t} + 0.319e^{-0.205t} + 0.330e^{-0.04t}$$

where t is in hours. Fernau and Smereker (1933) calculated a retention function for radon in the whole body of

$$R(t) = 1.183e^{-1.38t} - 0.183e^{-8.94t}$$

where t is in hours, while the results of Andersson and Nilsson (1964) suggest a single exponential retention function of

$$R(t) = e^{-1.4t}$$

yielding a removal half-time of 30 minutes. This retention function probably does not reflect the kinetics of the fat compartments.

The retention function for fat is less well established than that for the whole body. Hursh et al. (1964) calculated a retention function for fat of

$$R(t) = 0.00470e^{-0.036t} - 0.00313e^{-2.31t} - 0.00157e^{-0.23t},$$

where t is in hours. From their calculations, it follows that 70% of the decays from ingested radon after the first hour occur within the adipose tissue, with most of the decays in the first hour occurring in the stomach contents and the contents of the small intestine.

Hursh et al. (1965) employed their discerned retention functions to calculate the doses delivered to various body organs following ingestion of radon (not progeny). As indicated earlier, their calculations display a whole body dose equivalent (to non-stomach tissues) which is lower than that to the stomach by a factor of approximately 75 to 125. In addition, these authors calculated the dose to the lung from exhaled radon and concluded that this dose is small compared to that from radon dissolved in lung tissues.

No studies of age dependence have been published to date. Relative organ doses in rats, mice and adult humans, however, display great similarities, despite large differences in body mass. A probable explanation of this result lies in the relative invariance of the ratio of lung surface area to body mass for different ages and masses (Crawford 1980) and in the rapid equilibration of the radon in the body, which reduces the significance of age-dependent changes in G.I. tract segmental transport (Crawford 1983). This suggests that the equilibrium concentration of radon in body tissues and fat, as well as removal halftimes, are controlled by Ostwald coefficients, which do not change as a function of age or mass of the host. As a result, it is suggested that doses computed for adults following ingestion of a given activity of radon should apply at other ages following correction for body mass. In other words, doses at other ages will be inversely proportional to body mass. Corrections need be made, therefore, only for the age-dependent rate of water intake. In any event, organ doses and risks from inhaled radon significantly exceed those delivered following ingestion when radon-laden water is used in a dwelling. Since these calculations of ingestion dose have employed conservative assumptions, it is unlikely that more accurate knowledge of ingestion doses would affect regulatory decisions. The reader should bear in mind that the preceding two sentences assume that only the radon in the water, and not the existing progeny, are of regulatory concern and, hence, included in risk calculations.

Calculations of the dose from ingested radon are simplified by the fact that almost all of this dose arises from alpha decays. As a result, all of the decay energy may be assumed to be absorbed at the site of decay, avoiding the modeling problems associated with beta- and gamma-emitters. Letting C be the concentration of radon in a tissue of mass M, and letting E be the energy of an alpha decay, the dose, D, to a tissue between times t_1 and t_2 is

$$(6.24 \times 10^7)D = \int_{t_1}^{t_2} \underline{\lambda CE} \ dt,$$

where is the radiological decay rate constant for the radio active substance (minutes $^{-1}$), C is the concentration in units of atoms per gram of tissue, E is the alpha decay energy in units of MeV, and 6.24×10^7 is the MeV/g-rad conversion factor. The dose, D, is in units of rads (to the tissue of interest). For pertinent examples of this methodology, the reader is referred to the papers by Hursh et al. (1965) and Dundulis et al. (1984).

A simplified illustration of dose calculation may be developed by examining a calculation of the dose to the stomach wall which uses conservative assumptions of a single 45-minute removal half-time from the stomach (in line with the most recent work by Dundulis et al.) and immediate equilibration between the concentrations of radon in the stomach contents and in the stomach wall (Hursh 1965). Only the dose delivered by the ingested radon, and not by the ingested radon progeny, is considered, since only the radon is of regulatory concern here. In addition, only the decays within the wall are considered, since the contribution to the stomach wall dose from decays occurring in the contents is small. This is identical to assuming electronic equilibrium in the dose calculation.

Each radon decay in the body results in production of a RaA atom, followed, in turn, by RaB, RaC and RaC'. Assuming that no RaA, RaB, RaC and RaC' atoms leave the stomach wall before decaying, it may be seen that the total alpha energy released in the wall for each ^{222}Rn decay (see Figure II-3) is 5.49 + 6.00 + 7.69 = 19.18 MeV. Following the calculations of Hursh et al. (1965) and assuming an empty stomach content of 380 grams (200 grams of water, 130 grams of stomach mass and 50 grams of stomach digestive juices and other residuum), intake of 1 microcurie of ^{222}Rn in the water results in a stomach wall concentration of 1 microcurie/380 g, or 0.0026 microcurie/g. Since the retention function is a single exponential with a 45-minute half-time, the activity concentration, A, (microcurie/g) in the stomach wall at any time, t, following ingestion would be

$$A(t) = 0.0026e^{-0.693t/45}$$

From the previous equation, the integrated dose to the stomach wall from this 1-microcurie intake would be

$$D = \int_{0}^{\infty} \frac{(0.0026)(2.22 \times 10^{6})}{(6.24 \times 10^{7})} e^{-0.693t/45}(19.18)\ dt$$

$$= \frac{(1.77 \times 10^{-3})(45)}{(0.693)} = 1.15 \times 10^{-1} \text{ rad}$$

where 2.22×10^{-6} is the conversion factor from microcurie to disintegrations/minute. With quality factors of 10 and 20, this dose corresponds to dose equivalents of 1.15 and 2.3 rem, respectively. It will be noted that the conservative result is a factor of five above that calculated by Hursh et al. (1965). This difference is due to the shorter removal half-time (12 to 18 minutes) employed by the latter authors, to their neglect of the RaC decay energies and to their assumption that the ^{222}Rn is removed promptly from the stomach at one hour. Conservative calculations of the dose from ingestion of ^{222}Rn on a full stomach (680 g) yield dose equivalents of 642 and 1,285 mrem, assuming quality factors of 10 and 20, respectively.

Hursh et al. (1965) also calculated that 12% of the radioactive decays of ^{222}Rn which occur in the body following ingestion take place in the (non G.I. tract) soft tissues. Using a whole body retention half-time of 45 minutes indicates that approximately

$$\frac{0.693/(3.823)(24)(60)}{(0.693/45 + 0.693/[3.823][24][60])}$$

or 1% of the ingested radon decays somewhere in the body. For comparison, Hursh et al. (1965) found that 1.17% decays in the body. Using this upper value of 1.17%, it may be seen that 0.12×0.0117 or 0.14% of the ingested radon atoms decay in sites other than the G.I. tract. Using a mass of 2 kg for the G.I. tract, of 5kg for the skeletal mineral mass and of 70 kg for the total body, the mass of the "non G.I. tract" components of the soft body tissue is 63 kg. As a result, a conservative estimate of the average dose delivered to soft tissues from ingestion of 1 microcurie of ^{222}Rn is

$$D = \frac{(0.0014)(1)(2.22 \times 10^6)(19.18)}{(0.693/[3.823][24][60])(63000)(6.24 \times 10^7)}$$

or 1.3 mrem if a quality factor of 10 is employed or 2.6 mrem if a quality factor of 20 is employed. In the above equation, the factor

$$\frac{2.22 \times 10^6}{(0.693/[3.823][24][60]}$$

yields the number of atoms of ^{222}Rn present in one microcurie and 19.18 is the total energy of decay of radon plus progeny.

Hursh et al. (1965) also cited experimental results to determine that the kidney dose is higher than that to the average total body by a factor of 6 to 10, due to preferential translocation of RaC to the kidneys (only the progeny produced de novo were considered). This indicates a kidney dose/microcurie ^{222}Rn intake of 7.8 to 13 mrem if a quality factor of 10 is used and of 15.6 to 26 mrem if a quality factor of 20 is used (the dose calculated in the last equation has been employed here). The same authors concluded that 1.9% of the radon decays which occur in the systemic organs occur in the liver, a value which is slightly higher than that which would hold if radon were uniformly distributed. This raised value is due to the passage of blood directly from the G.I. tract to the liver before traveling to other organs. Using a liver mass of 1,700 g, intake of 1 microcurie of ^{222}Rn in water would result in a liver dose of

$$D = \frac{(0.019)(0.0117)(2.2 \times 10^{6})(19.18)}{([1700][0.693/3.823][24][60])(6.24 \times 10^{7})}$$

$$= 0.7 \text{ mrad}$$

A quality factor of 10 results in a dose equivalent of 7 mrem/microcurie intake, and a quality factor of 20 results in a liver dose equivalent of 14 mrem/microcurie intake. Again, ingestion of radon progeny has not been considered.

In summary, using conservative assumptions and a quality factor of 20, a reasonable upper limit on the dose equivalent delivered to the stomach would be 2.6 rem/microcurie intake of ^{222}Rn. This calculation assumes an empty stomach; the "full stomach" calculation yields 1.3 rem/microcurie intake of ^{222}Rn. These values are higher by a factor of two to five than previously cited calculations due to the longer (45-minute) half-time assumed for ^{222}Rn removal from the G.I. tract to the systemic organs and exhalation pathway. The dose equivalents to the kidney and liver are calculated to be less than 23 and 14 mrem/microcurie ^{222}Rn ingested, using a quality factor of 20. The dose equivalent averaged over the mass of soft tissue is similarly estimated to be less than 2.4 mrem according to the present calculations, with the dose equivalent to fat contributing most of this value. This is lower than that reported by Van Dobeln and Lindell (1964) by a factor of 1.7 and lower than that reported by Andersson and Nilsson (1964) by a factor of 5.8. Conservative estimates of the stomach (G.I. tract), liver, kidney and whole body dose equivalents from ingestion of 1 microcurie of ^{222}Rn would then be 1.8 (average of full and empty stomach values), 14, 24 and 10 mrem, respectively. This conservative estimate of the dose equivalent to the stomach lining is probably an overestimate by a factor of between two and ten. All calculations above have not included doses delivered through ingestion of radon progeny and do not include lung doses delivered by emanation of the radon from the stomach contents and into the lung passages. This latter component should be small.

C. Immersion Doses

A third component of exposures to radon and progeny following the use of radon-laden water in a dwelling concerns the external dose delivered by the radon and progeny present in the air of the dwelling. In this case, radiological concern is directed toward the photons and high-energy electrons emitted in decays, since these are much more penetrating than are the alpha particles which dominate internal exposures. The following calculations are based on the detailed report of Kocher (1981), who states:

> It is generally quite difficult to obtain realistic estimates of external dose from arbitrary distributions of radionuclides in the environment. Therefore, simplified and idealized exposure conditions are often assumed; namely, that the distribution of sources at any location in the environment is effectively infinite or semi-infinite in extent and the radionuclide concentrations are uniform throughout the source region. In addition,

it is often assumed that the only shielding between the radioactive sources and the body surface of the exposed individuals is provided by the medium through which the radiations are transmitted (i.e., air or water). Thus, no accounting is made for shielding provided by buildings during indoor residence, by clothing, or by buildings and irregular terrain during outdoor exposures.

For source regions which are assumed to be infinite or semi-infinite in extent with uniform radionuclide concentration, we are led to the concept of the external dose-rate conversion factor, which will usually be called the dose-rate factor. If a source region has uniform radionuclide concentration X at time t, the external dose rate D° for an exposed individual may be written in the general form

$$D^\circ(t) = X(t)\mathrm{DRF},$$

where DRF denotes the dose-rate factor defined by this equation as the external dose-rate per unit radionuclide concentration. The dose-rate factor thus embodies all aspects of the calculation of external dose-rate except for the radionuclide concentration.

Table III-2 displays values for the dose-rate factor for the radionuclides present in dwelling air following emanation of ^{222}Rn into the air. These values are assumed to apply to all age groups, since the factors do not change appreciably with age for photon exposures. The choice of these radionuclides follows directly from examination of Figure II-3, which displays the ^{222}Rn decay chain. It is assumed in this report that the exposure from radon progeny deposited on exposed surfaces of the dwelling is very small compared to the contribution from progeny entrained in the air.

Table III-2 Dose-Rate Conversion Factors for Immersion in Air Containing the ^{222}Rn Decay Chain (Sv/year per Bq/m^3)

Nuclide	Lung	Total body
^{222}Rn	4.59×10^{-4}	4.92×10^{-4}
^{218}Po	0.0	0.0
^{214}Pb	2.86×10^{-1}	3.08×10^{-1}
^{214}Bi	1.95	2.07
^{214}Po	1.04×10^{-4}	1.1×10^{-4}

From Kocher 1981. Includes alpha, beta and gamma decays and assumes uniform immersion in air containing the radionuclides. All radionuclides are assumed to be entrained in air. Alpha decay energies are assumed to be absorbed in the dead layer of the skin.

From this information, organ doses delivered by the entire decay chain may be easily estimated for any equilibrium factor and radon concentration. For example, assume that water use causes a concentration of 1 pCi/L of ^{222}Rn in dwelling air. From the present discussion of the ratio C_a/C_w, it may be noted that this corresponds to a water concentration of 10^4 pCi/L of ^{222}Rn. Also assume an equilibrium factor of 0.5, as described previously. This corresponds to a ^{218}Po concentration of 0.93 pCi/L; a ^{214}Pb concentration of 0.56 pCi/L, a ^{214}Bi concentration of 0.38 pCi/L and a ^{214}Po concentration of 0.38 pCi/L. These values may be obtained by reference to Table II-5. Using the dose-rate factors in Table III-2, these concentrations yield an annual dose equivalent of approximately 3.34 mrem/year to the total body and 3.12 mrem/year to the mass of the lung. By way of comparison, using the calculations in the Section on inhaled radon progeny, these same concentrations would yield an internal dose to the lung of 1.80 rem/year if an alpha quality factor of 10 is assumed and of 3.60 rem/year if a quality factor of 20 is assumed. Clearly, the inhalation dose from radon progeny entrained in the air is much larger than the external dose. As a result, the risk from radon released into the air from water need only include the contribution from internal irradiation of the lung tissues.

D. Comparison of Exposure Pathways

The results of the preceding three subsections may be summarized to provide a comparison of the doses imposed by the ingestion of ^{222}Rn in water, the inhalation of radon progeny in air and the exposure to external sources of the radionuclides in air. In this manner, it may be possible to focus attention on a single dominant pathway when considering the significance of epidemiological data on the predicted risk from use of radon-laden water in dwellings.

Calculations have already been presented which demonstrate that ingestion of water containing 10^4 pCi/L will result in an annual lung dose equivalent of 1.8 rem/year if an alpha quality factor of 10 is employed and of 3.6 rem/year if an alpha quality factor of 20 is employed. This was contrasted with the value of 3.12 mrem/year from external exposure, a value which is lower than that for inhalation by a factor of 1,000. As a result, calculations of the risk from radon released from water into the air need only focus on the contribution from inhalation of the progeny.

These values for lung (1.8 or 3.6 rem/year) may be compared with the dose equivalent delivered to the G.I. tract and to the total body as a result of direct ingestion of radon (but not progeny) in water. As noted in the Section on ingestion dosimetry, adult values for the stomach dose equivalent vary by as much as a factor of ten between the earlier and more recent work. As an upper limit to the internal doses delivered by ingested radon, the mean value of 600 mrem/microcurie of ^{222}Rn in water suggested by Cross et al. (1985) might be employed for the stomach dose. A concentration of 10^4 pCi/L would then translate to an annual intake of 1.1 microcurie of ^{222}Rn, assuming consumption of 300 mL of untreated water per day. This yields an annual dose equivalent of 660 mrem to the stomach. If the conservative value of 2.3 rem/microcurie intake, as calculated in this report, is used, the annual dose equivalent to the stomach is 2.5 rem. Similarly, use of the upper estimate for whole body (or, more correctly, total body) dose equivalent reported by (Cross et al. 1985) (10 mrem/microcurie of ^{222}Rn) would yield an estimated whole (total) body annual dose equivalent of 11 mrem. Both of these doses from ingestion would be lower if the most recent model by Dundulis et al. (1984) were employed. In this case, the stomach dose would be replaced by the dose to the G.I. tract and would drop to less than 100 mrem/year, with a value of approximately 4 to 6 mrem/year as an average for the remaining tissues. If the upper dose equivalent value for the stomach is employed (2.5 rem/year), the whole body effective dose equivalent (ICRP 77) would be 2.5 x 0.06 = 0.15 rem/year. This contrasts with the whole body effective dose equivalent of 3.6 x 0.12 = 0.43 rems/year from inhalation following use of the same water in the home for purposes other than drinking (such purposes dominate the use of water in homes). Even when the highest values for the stomach and whole body are assumed, the lifetime risk imposed by these pathways are factors of 5 and 500, respectively, below the lifetime risk imposed by the inhalation pathway (Cross 84). As a result, only the inhalation pathway need be considered of major importance in calculating risks from the use of radon-laden water in dwellings. This feature of the relative magnitudes of the risk from the three pathways should hold true at all ages of exposure.

IV. HUMAN EXPOSURES

A. Introduction

When the uranium miners of the 20th century began to display an elevated incidence of lung cancer, they became part of a chain of evidence linking radiation and lung cancer which extends back at least 500 years. The first clues to such a causal link were noted in the mining groups at Schneeberg, Saxony and Joachimsthal (now Jachymov), Bohemia. Between 44% and 75% (Weller 1965) of the metal miners in these groups developed a disease, labeled Bergkrankheit (miner's lung), characterized by coughing, mucoid, mucopurulent or bloody sputum, increasing dyspnea, loss of strength and weight, boring pain in the chest or back and, finally, death (Weller 1965). While he was not able to hypothesize a causal agent, it was clear to Agricola (the town physician of Joachimsthal in 1527) that the disease was connected in some manner to the conditions in the mines.

Two hundred and fifteen years would pass before the subject was again broached, this time by Scheffler (1770), who showed that the Bergkrankheit was not pneumonia, peripneumonia or asthma. It was another 100 years before Harting and Hesse (1879) could establish that the disorders resulted from a primary cancer of the lung, although they incorrectly labeled the cancers as lymphosarcomas. A number of possible etiological factors were proposed, including arsenic, cobalt, nickel, bismuth and silicon. These two physicians also initiated the current interest in the smoking habits of the study population.

The next 50 years witnessed a major shift in the paradigms of modern science, with a resultant influx of new experimental results (Kuhn 1970). Among the discoveries during this period was the determination that atoms could spontaneously emit radiations. These results came from the work of Roentgen in 1895 (x-rays) and Becquerel in 1896 (uranium). Although both scientists noted a reddening of the skin following exposure to these radiations, no theory existed to explain the interaction of the radiation with biological systems.

Studies of the lung cancers in the Schneeberg miners led Margarete Uhlig (1921) to propose, in 1921, that Bergkrankheit might arise from the inhalation of ^{222}Rn normally found in the mines. During this same year, Thiele et al. (1924) conducted a series of clinical, roentgenologic and pathologic studies of the miners. They concluded that the neoplasms were carcinomas, that they occurred only in persons who worked in the mines or were in intimate contact with the products and that the cancers were still endemic in the group. For an excellent review of these data, the reader is referred to the above references and to the compilation in Occupational Tumors and Related Diseases (Heuper 1942). A general conclusion is made evident by noting that approximately 75% of the deaths in the mining groups were a result of lung cancer; this indicates that the lung is the critical organ for inhalation of the radon decay chain.

Between 1923 and the present, a number of studies have been initiated to determine the concentration of radon and its radioactive progeny present in the various mines. Mining groups, including both uranium and hard rock miners, have been studied to determine the underlying form of the functional relation between lung cancers and exposure to radon. Among the study populations have been the Schneeberg miners (Sevc and Placek 1972), Joachimsthal miners (Pierquin et al. 1965), Canadian fluorspar miners (de Villiers and Windish 1964 and Parsons 1964), Colorado plateau uranium miners (Archer et al. 1973 and Archer 1976) and both uranium and non-uranium metal miners in the United States (Wagoner et al. 1963). These groups received relatively high doses to the lung. Low-dose studies among the uranium miners in the United States, Czechoslovakia and Canada and among the lead, zinc and iron miners in Sweden have been reviewed by Archer et al. (1980). Such studies typically reveal that lung cancer incidence increases with exposure to ^{222}Rn progeny in an approximately linear fashion, suggesting that the two might be viewed as cause and effect.

Until recently, interest in radon as a potential source of hazard continued to focus on exposures to ^{222}Rn progeny in the air of uranium and other mines. In the 1970s, however, measurements of ^{222}Rn and its progeny in non-industrial sites such as businesses and homes (Cliff 1978, Keller et al. 1982a,b, Steinhausler 1975, Stranden et al. 1979, Swedjemark 1978a UNSCEAR 1982, Nero 1983a, Nero 1983b and Gesell 1983 and NCRP 1984a) suggested that significant concentrations of the progeny could exist in more normal settings. For example, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 1982) has reviewed the available data and estimates that the average radon concentration in outdoor air is approximately 3 Bq/m^3 (1 Bq is equal to one disintegration per second, or 27 pCi). This assumes an equilibrium factor (defined later) of 0.6, thereby yielding an estimated equilibrium equivalent concentration of 1.8 Bq/m^3.

Levels of radon and progeny outdoors are typically lower than those indoors. Indoor concentrations are raised because of the decreased ventilation rate, which results in a smaller volume of air into which emanated radon may be diluted. Factors affecting the build-up of radon and progeny in buildings will be discussed in a later section. For purposes of this Introduction, however, it may be noted that UNSCEAR (1982) determined that the average equilibrium equivalent concentration of ^{222}Rn and progeny in homes located in temperate regions of the world is approximately 15 Bq/m^3, with a wide range of values in different countries extending from 5 to 60 Bq/m^3 (0.14 to 1.62 pCi/L). They estimate that the average annual effective dose equivalent resulting from both indoor and outdoor exposures to ^{222}Rn and progeny is 1 mSv (100 mrem) per year, (to the lung) which is equal to the combined background radiation from all other sources. The data from the U.N. report (UNSCEAR 1982) are reproduced in Tables II-2 and IV-1 for reference. In a later section, it will be shown that the UNSCEAR values here are significantly lower than current measured values in the United States.

The figures above suggest that the presence of ^{222}Rn and progeny in homes might provide a significant source for exposure of the general public to radiation. Certainly, a large component of this emanation arises from the presence of ^{226}Ra in the soil and building materials surrounding the location of interest. Measurements of ^{222}Rn dissolved in water used by homes and offices, however, (Hess et al. 1982) indicate that the use of such water in homes could provide another significant source of ^{222}Rn emanation into living and working spaces. The emanation of the partially dissolved radon from water to air in homes has been established by several experimental studies (Partridge et al. 1979, Gesell and Prichard 1980, Hess et al. 1981, 1982) and confirms that appreciable quantities of the radon in water may enter the air in which the water use occurs.

Table IV-1. Estimated per capita Annual Effective Dose Equivalents From Natural Sources in Areas of Normal Background

Source of irradiation	Annual effective dose equivalent (microsieverts) External irradiation	 Internal irradiation	 Total
Cosmic rays			
Ionizing component	280		280
Neutron component	21		21
Cosmogenic radionuclides		15	15
Primordial radionuclides			
^{40}K	120	180	300
^{87}Rb		6	6
^{238}U series			
$^{238}U \rightarrow {}^{234}U$	90	10	1,400
^{230}Th		7	
^{226}Ra		7	
$^{222}Rn \rightarrow {}^{214}Po$		800	
$^{210}Pb \rightarrow {}^{210}Po$		130	
^{232}Th series			
^{232}Th	140	3	326
$^{228}Ra \rightarrow {}^{224}Ra$		13	
$^{220}Rn \rightarrow {}^{208}Tl$		170	
Total (rounded)	650	1,340	2,000

Adapted from UNSCEAR (1982).

B. Radon Concentrations in Drinking Water

A previous section introduced the basic physical concepts necessary to define the extent to which airborne radon progeny and emanated radon contribute to human exposures. Before turning to the calculation of the doses and risks associated with various levels of radon exposure, it is necessary to document that radon dissolved in water does, in fact, appear in significant quantities in water supplies. Having documented the levels of ^{222}Rn in water in the United States and the extent to which this radon may enter the indoor air, the present report will return to the previous considerations in estimating associated health risks to the U.S. population.

Much of the information reported in this section was obtained from the document prepared by Hess et al. (1985) on the occurrence of ^{222}Rn in public water supplies in the United States. Those authors reviewed existing literature on ^{222}Rn concentrations in various water supplies located throughout the world and in the continental United States.

As mentioned earlier, radon is an inert gas that is soluble in water. Its source of production lies in the ^{226}Ra found in soil and rocks. The concentration of ^{226}Ra and hence ^{222}Rn, is highest in granitic rocks (Stranden 1981), uranium minerals, such as uraninite or carnotite (Taniguchi and Vasudev 1980), and the waste products of phosphate fertilizer production facilities (USEPA 1979). The tailings piles from uranium mines, which are high in ^{226}Ra content are an additional source. The translocation of the radon and its subsequent movement into water are determined by the geochemical properties of the ^{226}Ra and host soil or rock, the physical condition of the rock matrix (fractures, etc.) and environmental factors such as pressure, temperature and radon emissivity from the host material. The mechanisms by which the radon is released from the host material and translocated through an aquifer, as well as controlling factors, have been reported by several investigators (Andrews and Woods 1972, Rama 1983, Tanner 1964, Taniguchi and Vasudev 1980). Results indicate that movement through rock and soil is dominated by diffusion along microcrystalline imperfections in the host matrix. Once the radon enters groundwater, the transport becomes dominated by direct water transport, characterized by percolation velocities greater than 10^{-5} cm/sec (Hess et al. 1983).

The previously mentioned study on occurrence (Hess et al. 1985) summarized the variation of radon content with rock types for well water in Maine. Their summary is reproduced in Table IV-2. They noted that radon concentrations in water may differ by a factor of 20 depending on the rock types. Low radon concentrations appeared to be associated with public utility water stored in gravel aquifers, in which greater

Table IV-2 Average Radon Values in Individual Water Systems and Public Water Supplies

	Arithmetic mean (pCi/L)	Maximum (pCi/L)	Number of samples
Maine (Hess 1979, Hess et al. 1983b)	10,000	1,000,000	2,000
In granite zones	22,000	300,000	136[a]
In sillimanite grade zone	13,600	100,000	35[a]
In chlorite	1,100	2,500	56[a]
In public utilities	2,000	11,700	64

[a] Rock grade determined by geologist for each private well.

Adapted from Hess et al. (1985).

quantities of water are available for the dilution of the emanated radon. Even lower concentrations were found in surface water.

Radon concentrations in water have been measured for a range of sources world wide, including groundwater, well water, tap water, rain water, oceans, rivers and thermal spas. Reported measurements were performed either in response to radiological needs or in attempts to test new methods for assessing the ^{222}Rn concentration in water samples. These measurements have been documented in the United States (Arndt and Kuroda 1953, Fresco 1955, Gesell 1977, Gesell and Prichard 1980, Hess et al. 1980, Kuroda et al. 1954, Placak and Morton 1950, Prichard 1978, Prichard and Gesell 1981, Rogers 1958, Sasser and Watson 1978, Schlundt 1935, Smith et al. 1961, Strain et al. 1979, Tanner 1964), Finland (Asikainen and Kahlos 1979, Asikainen and Kahlos 1980, Castren et al. 1977, Castren 1980, Kahlos 1969, Kahlos and Asikainen 1973, Kahlos and Asikainen 1980, Kahlos and Suomela 1970, Suomela and Kahlos 1972), Japan (Kuroda and Yokoyama 1948, Miyake et al. 1980), Israel (Mazor 1962), Sweden (Dobelin and Lindell 1964, Snihs 1973), Germany (Muth et al. 1960), India (Kelkar and Joshi 1969), Canada (Simpson et al. 1959), Yugoslavia (Kobal and Kristan 1972, Miholic 1952), New Zealand (Belin 1959), England (Abbatt et al. 1960, Hoather and Rackham 1963, Jacobi 1949, Kenny et al. 1966, Turner et al. 1961), France (Curie and Laborde 1904), Austria (Steinhausler and Pohl 1972, Uzunov et al. 1981) and the Netherlands (Hoogteijling et al. 1948, Sizoo and Koene 1938). In general, concentrations of ^{222}Rn in accessible groundwater supplies tended to average less than 10,000 to 20,000 pCi/L, but values higher than 100,000 pCi/L were not uncommon, with the highest concentrations being in drilled wells. A summary of the available literature is contained in Table IV-3. (For a complete discussion of the most recent data, the reader is referred to Hess et al. 1985 or to NCRP 1984b.)

The present report focuses on the concentration of radon in water supplies in the United States, and the data in accompanying Figures and Tables obtained from Hess et al. 1985 A listing of radon concentrations in different states is presented in Table IV-4, arranged by state and source of water. Table IV-5 presents similar information, arranged by the size of the population using a given water supply. The mean radon concentration in public groundwater supplies is displayed in Figure IV-1, and a histogram of similar information is contained in Figure IV-2. A blind sampling of public water supplies in the central United States resulted in the information contained in Table IV-6. While the latter data possess the desirable characteristic of being a more random sample than those in previous tables, the data themselves are less numerous.

The above information suggests that the radon concentration in public waters is higher than that of uranium or radium

Table IV-3 Summary of the Information on ^{222}Rn Concentrations in Water

Reference[a]	Country	Source	Mean (pCi/L)	Maximum (pCi/L)
Abbatt et al. (1960)	England	Various	α^b	13,300
Arndt and Kuroda (1953)	U.S., Arkansas	Creeks, streams, lakes ground water	α 59,000	3,160 α
Asikainen and Kahlos (1979)	Finland	Tap - drilled - dug	55,000 2,600	880,000
Asikainen and Kahlos (1980)	Finland	Tap	670	17,000
Belin (1959)	New Zealand	Thermal spring condensate	≈80,000	250,000
Castren (1980)	Finland	Bored wells - Helsinki - other parts	40,000 8,000	1,200,000
Dobeln and Lindell (1964)	Sweden	Tap	150	33,000
Frank (1955)	U.S., New York	Deep water	≈12,000	24,600
Gesell and Prichard (1980)	U.S., Florida	Tap - private wells - city supply	6,000 90	α

continued -

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[a] References which contain only measurements originally reported elsewhere are not shown.
[b] = not reported.

Table IV-3 - continued

Reference[a]	Country	Source	Mean (pCi/L)	Maximum (pCi/L)
Gesell and Prichard (1980)	U.S., Houston	Tap	1,600	2,000
Hess et. al. (1980)	U.S., Maine	Tap	10,000	200,000
Hoather and Rackham (1963)	England	Boreholes	15,000	α
Hoogteijling et al. (1948)	Netherlands	Shallow groundwater	α	173
Jacobi (1949)	England	Tap	α	31
Kahlos and Asikainen (1980)	Finland	Tap - utilities - dug wells - drilled wells	670 1,600 17,000	 1,200,000
Kelkar and Joshi (1969)	India	Thermal springs	≈400	985
Kenny et al. (1966)	England	Tap	≈3,000	20,000

continued -

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[a] References which contain only measurements originally reported elsewhere are not shown.
b = not reported.

Table IV-3 - continued

Reference[a]	Country	Source	Mean (pCi/L)	Maximum (pCi/L)
Kobal and Kristan (1972)	Yugoslavia	Thermal springs	450	970
Kuroda et al. (1954)	U.S., Arkansas	Thermal springs	α^b	30,000
Mazor (1962)	Israel	Various	≈1,500	21,000
Miyake et al. (1980)	Tokyo	Rain water	≈0.2	0.53
Muth et al. (1960)	Germany	Tap	200	α
Placak and Morton (1950)	U.S., various	Springs	α	260,000
Prichard (1978)	U.S., Texas	Tap	≈3,500	α
Prichard and Gesell (1981)	U.S., Houston	Tap	437	α
Rogers (1958)	U.S., Utah	Streams Springs	α α	197 3,100

continued -

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[a] References which contain only measurements originally reported elsewhere are not shown.
b = not reported.

Table IV-3 - continued

Reference[a]	Country	Source	Mean (pCi/L)	Maximum (pCi/L)
Sasser and Watson (1978)	U.S., N. Carolina	Tap	≈1,500	46,600
Simpson et al. (1959)	Canada	Mine	α	100,000
Sizoo and Koene (1938)	U.S., Maine New Hampshire	Drilled wells Dug wells	50,000 7,000	300,000 31,700
Strain et al. (1979)	U.S., N. Carolina	Tap	2,000	19,000
Tanner (1964)	U.S., Utah	Tap	700	1,800
Turner et al. (1961)	England	Tap	α	10,000
Uzunov et al. (1981)	Austria	Spas	40,000	α

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[a] References which contain only measurements originally reported elsewhere are not shown.
[b] = not reported.

Table IV-4 Radon in Water Results by State and Source[a]

State	Private well	Public water supply[b]	Public ground-water supply	Public surface water supply
AL	120 (22)	8 (31)	70 (182)	ND[c] (8)
AR	230 (2)	1,400 (1)	12 (22)	ND (1)
AZ	--	--	250 (124)	ND (6)
CA	43 (6)	790 (2)	470 (15)	ND (2)
CO	--	--	230 (76)	--
DE	--	--	30 (72)	--
FL	6,000 (34)	320 (2)	30 (327)	--
GA	2,100 (2)	44 (32)	67 (225)	43 (2)
IA	--	--	220 (85)	ND (2)
ID	--	--	99 (155)	--
IL	--	--	95 (314)	--
IN	--	--	35 (185)	--
KS	--	--	120 (47)	74 (2)
KY	1,500 (10)	ND (18)	32 (104)	ND (5)
MA	1,000 (8)	7 (2)	500 (212)	38 (2)
ME	7,000 (24)	990 (71)	--	--
MN	1,400 (1)	600 (1)	130 (233)	--
MO	ND (2)	--	24 (138)	ND (2)
MS	--	260 (2)	23 (104)	--
MT	4,300 (8)	--	230 (71)	ND (6)
NC	15 (29)	27 (2)	79 (404)	ND (4)
ND	--	440 (2)	35 (133)	--
NH	1,400 (18)	9 (12)	940 (52)	ND (6)
NJ	--	--	300 (38)	--
NM	59 (14)	45 (8)	55 (171)	ND (18)
NV	--	--	190 (57)	--
NY	1,500 (4)	34 (20)	52 (292)	ND (1)
OH	--	--	79 (165)	--
OK	--	--	93 (83)	--
OR	450 (18)	--	120 (69)	ND (4)
PA	910 (16)	--	380 (105)	--
RI	6,500 (69)	5,200 (6)	2,400 (575)	ND (10)
SC	1,100 (28)	--	130 (384)	ND (14)
SD	4,200 (2)	59 (2)	210 (155)	--
TN	ND (2)	ND (2)	12 (98)	--
UT	--	--	150 (195)	--
VA	560 (42)	--	350 (284)	ND (4)
VT	210 (23)	840 (4)	660 (71)	13 (16)
WI	730 (40)	28 (4)	150 (278)	ND (12)
WY	--	--	330 (32)	ND (2)
US	920 (434)	68 (224)	130(6,298)	1 (131)

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[a] Results are geometric means in units of pCi/L. Values in parentheses are numbers of samples. (From Hess 1985)

[b] May include both ground and surface water.

[c] ND = not detected above background levels.

IV-5 Radon in Water Results by State and Population

From Hess 1985.

STATE	Population Ranges											
	< 100		100-1,000		1,000-5,000		5,000-10,000		> 10,000		Unknown	
AL	--		83	(10)[b]	59	(76)	82	(46)	68	(44)	170	(6)
AR	--		32	(6)	6	(3)	--		12	(2)	8	(13)
AZ	--		240	(2)	200	(68)	350	(22)	340	(30)	160	(2)
CA	--		--		--		--		--		470	(15)
CO	--		130	(6)	220	(54)	400	(8)	300	(8)	--	
DE	--		100	(4)	39	(44)	11	(12)	23	(12)	--	
FL	--		320	(2)	290	(4)	49	(78)	24	(243)	--	
GA	57	(4)	42	(12)	130	(56)	190	(27)	52	(45)	39	(81)
IA	--		1,120	(13)	230	(36)	150	(24)	66	(10)	75	(2)
ID	6	(3)	210	(14)	130	(83)	36	(25)	100	(30)	--	
IL	--		71	(1)	81	(30)	80	(98)	110	(185)	--	
IN	--		--		45	(71)	17	(58)	50	(56)	--	
KS	260	(2)	230	(12)	43	(11)	320	(4)	58	(8)	210	(10)
KY	--		20	(10)	42	(76)	36	(8)	5	(10)	--	
MA	3,300	(2)	--		380	(47)	540	(67)	510	(88)	580	(8)
ME	670	(3)	1,600	(23)	690	(33)	2,700	(7)	450	(4)	400	(1)
MN	--		180	(22)	150	(76)	68	(43)	140	(90)	330	(2)
MO	ND[c]	(2)	58	(54)	26	(54)	1,100	(4)	78	(6)	ND	(18)
MS	--		--		15	(45)	51	(26)	23	(33)	--	
MT	740	(4)	280	(6)	270	(49)	160	(8)	18	(4)	--	
NC	11,000	(6)	250	(111)	45	(229)	16	(32)	21	(16)	8,100	(10)
ND	--		13	(10)	39	(112)	97	(5)	10	(6)	--	
NH	1,700	(2)	1,200	(4)	960	(24)	1,000	(14)	550	(8)	--	
NJ	--		--		--		360	(6)	1,200	(12)	120	(20)
NM	1,300	(2)	620	(7)	48	(89)	42	(16)	33	(49)	450	(8)
NV	--		220	(2)	240	(36)	72	(9)	530	(2)	180	(8)
NY	--		--		56	(59)	31	(85)	71	(113)	56	(35)

continued -

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[a] All results are geometric means in units of pCi/L. Values in parentheses are in numbers of samples.
[b] Number of data points used to calculate geometric mean.
[c] ND - not detected above background levels.

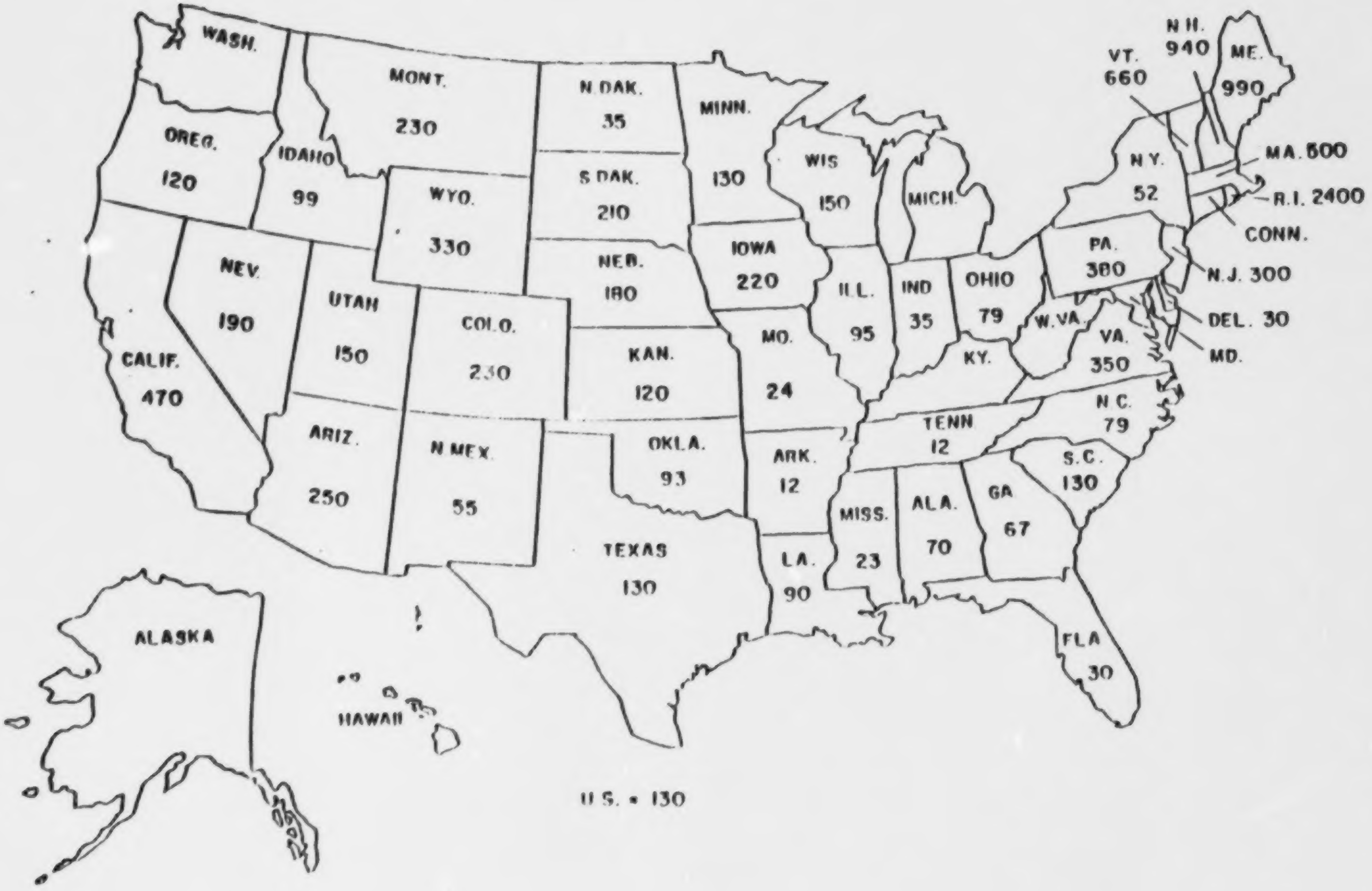
IV-5 - continued

	Population Ranges					
STATE	< 100	100-1,000	1,000-5,000	5,000-10,000	> 10,000	Unknown
OH	--	300 (2)	160 (4)	56 (76)	100 (83)	--
OK	--	260 (1)	65 (33)	190 (21)	79 (26)	96 (2)
OR	--	210 (2)	110 (48)	23 (6)	320 (6)	180 (7)
PA	1,900 (2)	--	260 (34)	450 (32)	440 (37)	910 (16)
RI	1,700 (91)	3,900 (325)	980 (71)	1,300 (30)	1,200 (58)	--
SC	1,400 (32)	870 (30)	92 (229)	74 (60)	60 (31)	410 (2)
SD	310 (16)	300 (41)	160 (85)	210 (8)	200 (5)	--
TN	--	160 (4)	19 (48)	6 (25)	5 (21)	--
UT	--	260 (2)	140 (104)	200 (39)	170 (48)	150 (2)
VA	85 (6)	880 (56)	140 (151)	320 (12)	720 (14)	2,300 (45)
VT	120 (1)	--	540 (24)	--	1,000 (2)	750 (44)
WI	--	--	150 (169)	190 (61)	130 (48)	--
WY	--	880 (6)	230 (19)	530 (6)	54 (1)	--
US	990 (175)	620 (777)	98(2,446)	92(1,098)	88(1,464)	140 (338)

[a] All results are geometric means in units of pCi/L. Values in parentheses are in numbers of samples.

[b] Number of data points used to calculate geometric mean.

[c] ND = not detected above background levels.


WASH.
OREG.
120
CALIF.
470
NEV.
190
IDAHO
99
MONT.
230
WYO.
330
UTAH
150
ARIZ.
250
COLO.
230
N.MEX.
55
N.DAK.
35
S.DAK.
210
NEB.
180
KAN.
120
OKLA.
93
TEXAS
130
MINN.
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IOWA
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MO.
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90
WIS
150
ILL.
95
MISS.
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MICH.
IND
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OHIO
79
KY.
TENN
12
ALA.
70
GA
67
FLA
30
S.C.
130
N.C.
79
VA.
350
W.VA.
PA.
380
N.Y.
52
VT.
660
N.H.
940
ME.
990
MA.500
R.I. 2400
CONN.
N.J. 300
DEL. 30
MD.
ALASKA
HAWAII
U.S. = 130

Figure IV-1 Geometric Average Radon Concentration in Public Groundwater Supplies (pCi/L)

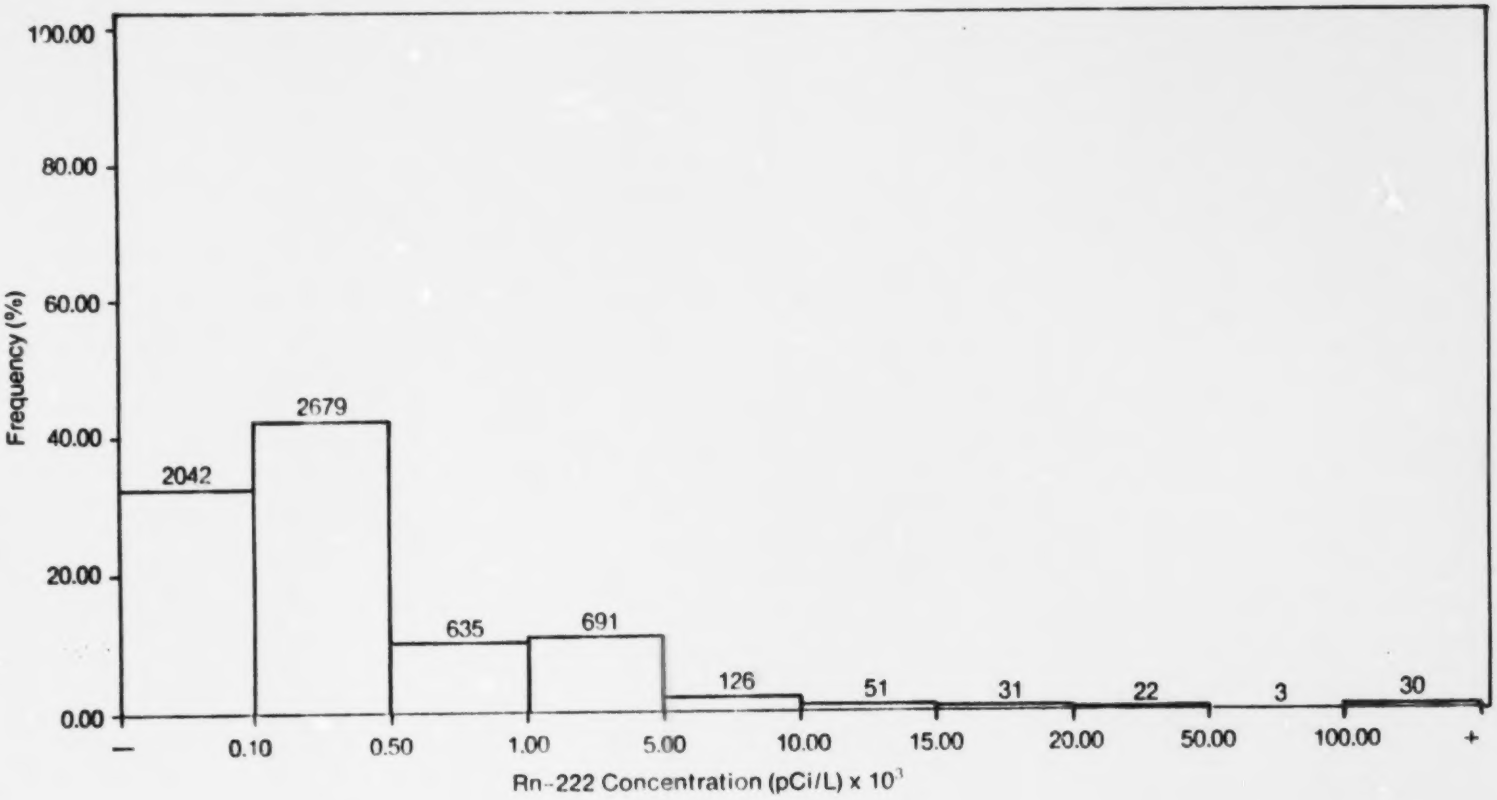


Figure IV-2 Public Groundwater Supplies (All)

Table IV-6 The Distribution of Radon in Municipal Water Supplies in the Central United States

State	Well waters			Distribution systems		
	N	G.M. (pCi/L)	G.S.D.	N	G.M. (pCi/L)	G.S.D.
Arkansas	6	135	2.66	20	47	1.95
Indiana	10	151	2.13	23	70	2.20
Iowa	33	175	2.13	31	111	2.45
Louisiana	61	151	1.84	22	93	2.54
Minnesota	28	252	2.08	28	183	2.65
Nebraska	47	262	1.85	21	178	3.68
New Mexico	27	287	2.39	20	220	2.16
Oklahoma	7	117	1.74	6	134	1.17
Composite	209	197	2.10	174	115	2.75
Texas				278	131	2.70

G.M. = Geometric Mean

G.S.D. = Geometric Standard Deviation

by a factor of several thousand (Hess et al. 1985). This is due to the increased mobility of the radon relative to either the uranium or the radium in the host rock (King et al. 1982). The highest radon water concentration was found in the Appalachian Mountain region, including the states of Rhode Island, Maine, New Hampshire, Vermont, Massachusetts, Pennsylvania and Virginia. Within those states, the highest radon values were associated with the granitic regions of the Appalachian Highlands Piedmont Provinces (Figure IV-3). Concentrations decreased in the midwestern and coastal plain and increased again when the mountain states of the west were sampled. Values for private groundwater supplies in the United States are displayed in Table IV-2, which indicates that the concentration of radon in private supplies is higher than that associated with public groundwater supplies by a factor of 3 to 20 (Hess et al. 1985). Presumably, this results from the use of low-capacity wells for the private supplies, whereas the public supplies use high-capacity sand or gravel wells. In general, surface supplies have much lower concentrations of ^{222}Rn, typically ranging below 100 pCi/L (Hess et al. 1983), which are probably due to increased aeration of the water and to the large surface area available for radon emanation into the air.

The most recent estimate of the geometric population average for radon in water in the United States is 187 pCi/L (Hess et al. 1985). The data contained in the previous tables

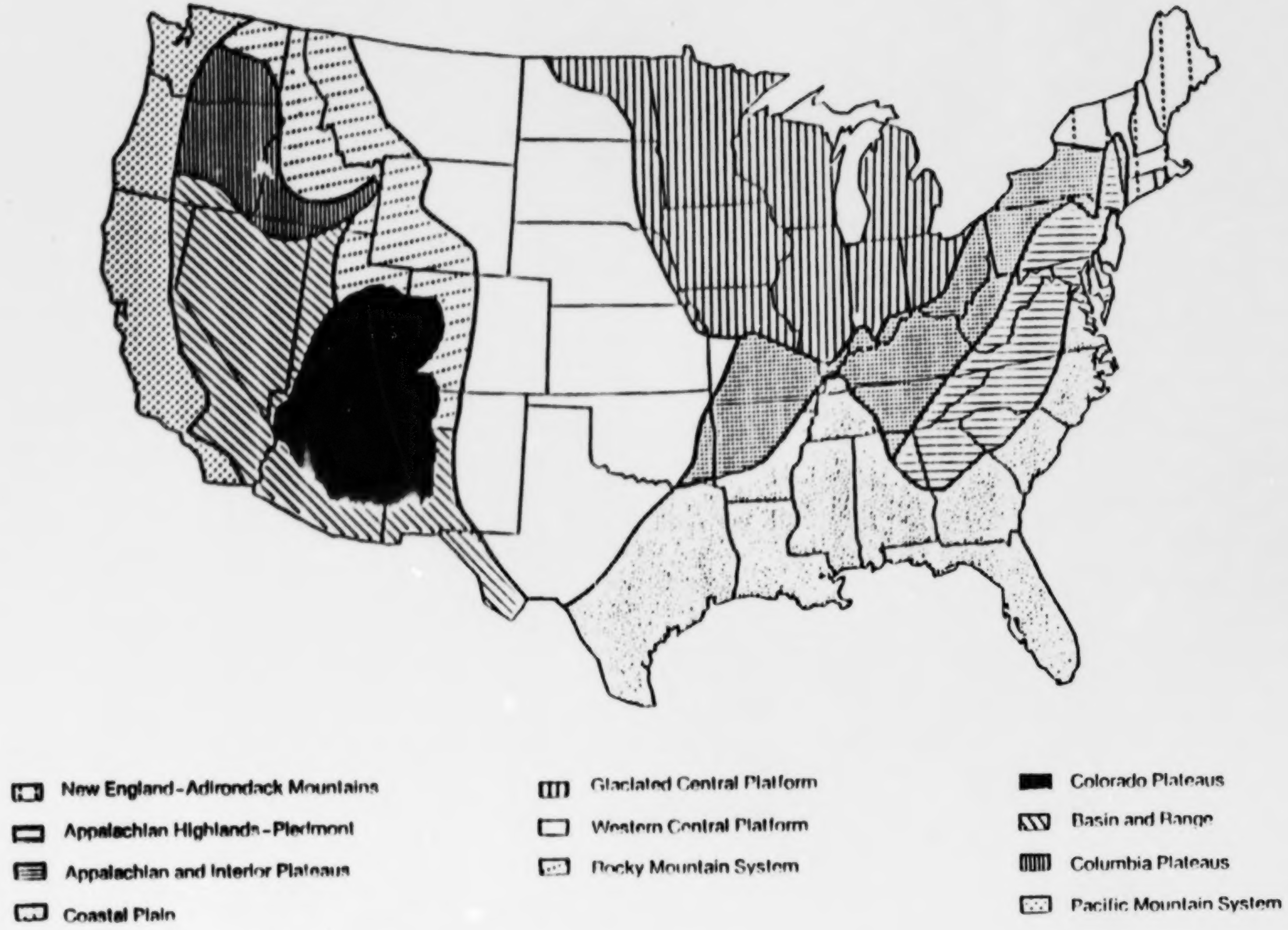

▭ New England-Adirondack Mountains
▭ Appalachian Highlands-Piedmont
▤ Appalachian and Interior Plateaus
▭ Coastal Plain
▥ Glaciated Central Platform
☐ Western Central Platform
▭ Rocky Mountain System
■ Colorado Plateaus
▧ Basin and Range
▥ Columbia Plateaus
▭ Pacific Mountain System

Figure IV-3 Mineral Regions in the United States

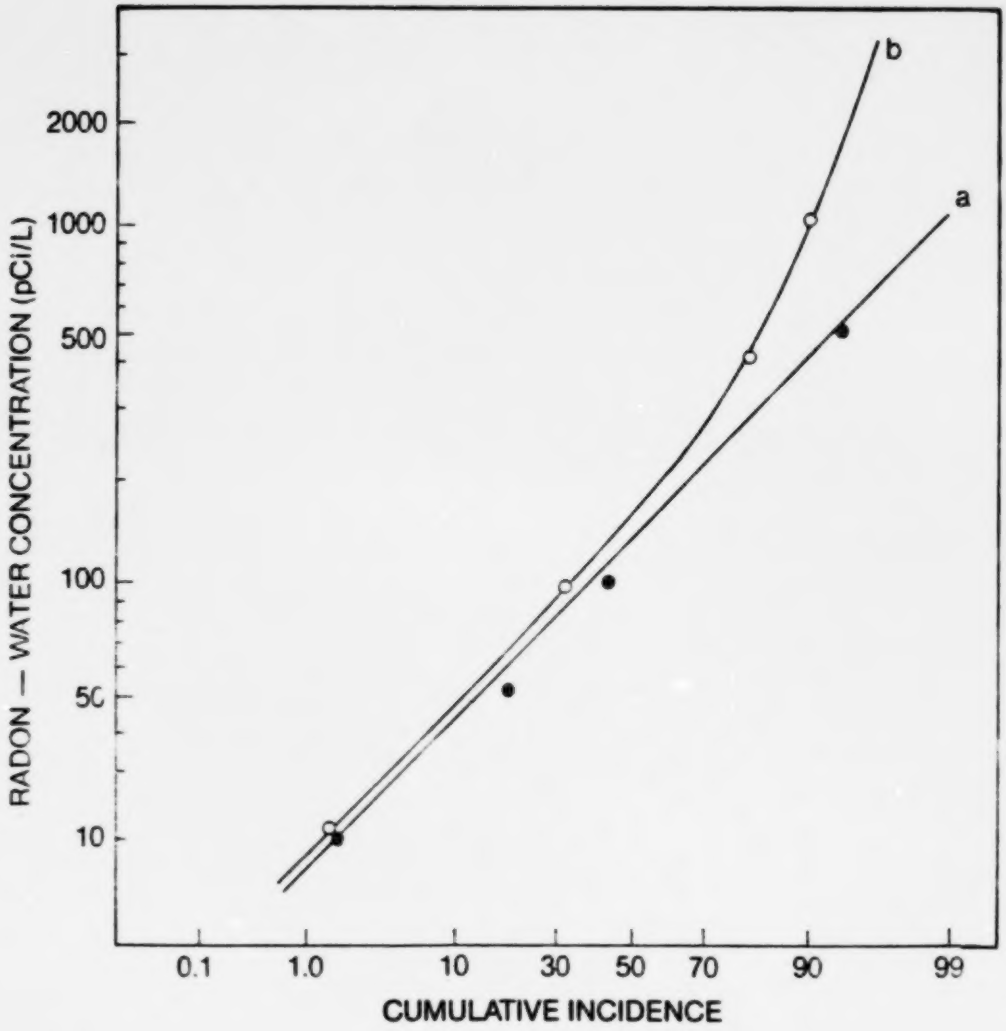


Figure IV-4 a) Distribution of Population in the United States Using Radon-Laden Public Groundwater Supplies
b) Distribution of Radon Concentrations in Public Groundwater Supplies

have been combined in Figure IV-4, which displays the percentage of the U.S. population using a groundwater supply that exceeds any given of the concentration. Figure IV-4 was generated through use of the data in Table IV-7, assuming that each category of water source was characterized by a log normal distribution throughout the United States, with a geometric standard deviation of 3.0 (the 6 S.D. associated with well waters and distribution systems in Table IV-6).

C. Translocation of Radon from Water to Air

This section discusses the extent to which radon may be emanated from water into the air of a structure and the mechanisms by which this process occurs. In addition, the results of empirical determinations of the water-to-air transfer fraction are presented.

In general, gases such as radon move along concentration gradients, with net movement occurring from sources or regions of higher concentration (in this case, potable water) to regions of lower concentration (air in a dwelling). The process by which this occurs is termed mass transfer, with further subdivision into components of molecular mass transfer and convective transfer. Molecular mass transfer refers to the propensity of molecules in a liquid or gas to undergo a "random walk" through the surrounding material, resulting in transport of the molecule to regions other than its initial

Table IV-7 Distribution of Radon in United States Groundwater Supplies

Population	Number of utilities samples	Mean concentration of radon in water (pCi/L)	U.S. population using this water (millions)
		GEOM - ARITH	
< 100	88	990 - 6,500	1.03
100 - 1,000	377	620 - 4,100	7.4
1,000 - 5,000	1,223	98 - 390	14.0
5,000 - 10,000	549	92 - 350	8.4
10,000 - 100,000	704	92 - 290	28.3
> 100,000	32	52 - 150	14.3
			73.43

---

[a] Population-weighted average = 187 pCi/L - 944 pCi/L.

location. Convective transfer refers to the movement of a molecule as determined by the bulk flow of surrounding material. In effect, the molecule of interest is carried by the flow of the host material.

Both processes operate on the radon atoms present in a body of water introduced into a dwelling. The concentration of radon in household air is typically lower than that in the water, creating a concentration gradient that results in radon being emanated from the water to the air. This emanation occurs in two distinct stages. The first stage consists of molecular mass transfer to the air-water interface, which is followed by convective transport (and some further molecular transport) into the surrounding air. Once introduced into the air, the radon tends to distribute fairly uniformly throughout any given room. From these considerations, it may be deduced that radon will be most efficiently transferred from water-to-air when the ratio of water surface area to volume is large.

The use of radon-laden potable water in a dwelling, therefore, may be expected to raise the radon concentration in the air of the dwelling. The extent to which the air radon concentration is raised will depend upon the quantity of water used and the water-to-air transfer efficiencies associated with the various uses. Some typical household uses of potable water include showers, baths, toilets, dishwashers, laundry facilities, cooking and use of water in sinks.

Table IV-8 reports measured values of the water-to-air transfer efficiencies for a number of household water uses. The transfer efficiency is defined as the fraction of radon emanated from the water into the surrounding air. From these data, it will be noted that radon transfer is most efficient when the water is heated and/or dispersed into droplets. Heating effectively raises the diffusion velocity of radon atoms in the water, bringing the atoms to the water surface more rapidly and facilitating their emission from the surface. Heated water, therefore, shows higher transfer efficiencies per unit water surface area. Dispersing the water, as occurs in baths, showers or dishwashing, increases the surface area of the water/air interface.

Gesell and Prichard (1980) have studied empirically the effects of heating on radon emanation from water, and their results are displayed graphically in Figures IV-5 and IV-6. From Figure IV-5, it may be noted that the boiling of water results in very rapid and efficient transfer of radon from water to the air. The data of Gesell and Prichard (1980) was obtained by keeping the water/air interface surface area constant and by minimizing the agitation of the water samples. It should also be noted that the data in Figure IV-6 indicate that the transfer efficiency also depends strongly on the amount of time a water source remains in a home. In addition, Gesell and Prichard (1980) measured the effects of agitation on radon loss from water. When water was agitated at a constant

Table IV-8 Measured ^{222}Rn Water-Air Transfer Efficiencies For Typical Household Activities

	Transfer efficiency (% ^{222}Rn released)				
	EPA 1977	Partridge 1979	Gesell 1980	Hess 1981	Hess 1982
Laundry washing					
Hot wash cycle (18 min) with soap		98.4±1.3			
Hot wash cycle (13 min) without soap		97.9±2.7			
Cold wash cycle (18 min) with soap		93.3±5.2			
Cold wash cycle (18 min) without soap		93.5±3.4			
Warm wash cycle (18 min) with soap		98.3			
Cold wash cycle (11 min) with soap		91.4			
Cold wash cycle (4 min) with soap		84.7			
Cold wash gentle cycle with soap		78.7			
Cold wash gentle cycle without soap		76.6			
Cold rinse regular cycle		80.9±17.4			
Cold rinse gentle cycle		62.2			
No specific description given			90[a]	90[a]	90[a]
Dishwasher					
Wash cycle		97.7±3.7			
Rinse cycle		98.5±2.1			
No specific description given			98	98	98
Bath Tub					
Hot water		59.7			
Warm water		36.2			
Cold water		37.8			
No specific description given			47	30[a]	30[a]
Shower					
Warm water		71.2±4.7			
No specific description given	91		63	65	65

continued -

Table IV-8 - continued

	Transfer efficiency (% ^{222}Rn released)				
	EPA 1977	Partridge 1979	Gesell 1980	Hess 1981	Hess 1982
Sink					
Warm water		28.3			
No specific description given				30[a]	10-15[a]
Toilets					
Tank		4.9			
Bowl		23.6±6.5			
No specific description given			30	30[a]	30[a]
Drinking and kitchen					
No specific description given			30		
Cleaning					
No specific description given			90		
Overall weighted average for all household uses		62.5	52	59	59

[a] Estimated.

room temperature, 40% of the radon was emanated in 15 minutes.

From Table IV-8, it may be noted that a typical average value for the radon transfer efficiency from water to air, weighted by the relative use of water in different activities, is in the region of 50% to 60%. An average transfer efficiency of 55%, therefore, is likely to be a good estimate of the average in the United States and is employed as a baseline in the present report.

Both Gesell and Prichard (1980) and Hess et al. (1982) have developed the steady state equation that relates radon concentration in potable water to the concentration of radon in dwelling air. Their equation is:

$$C_a = (C_w)(F)(V_w)/(\lambda)(V_h)$$

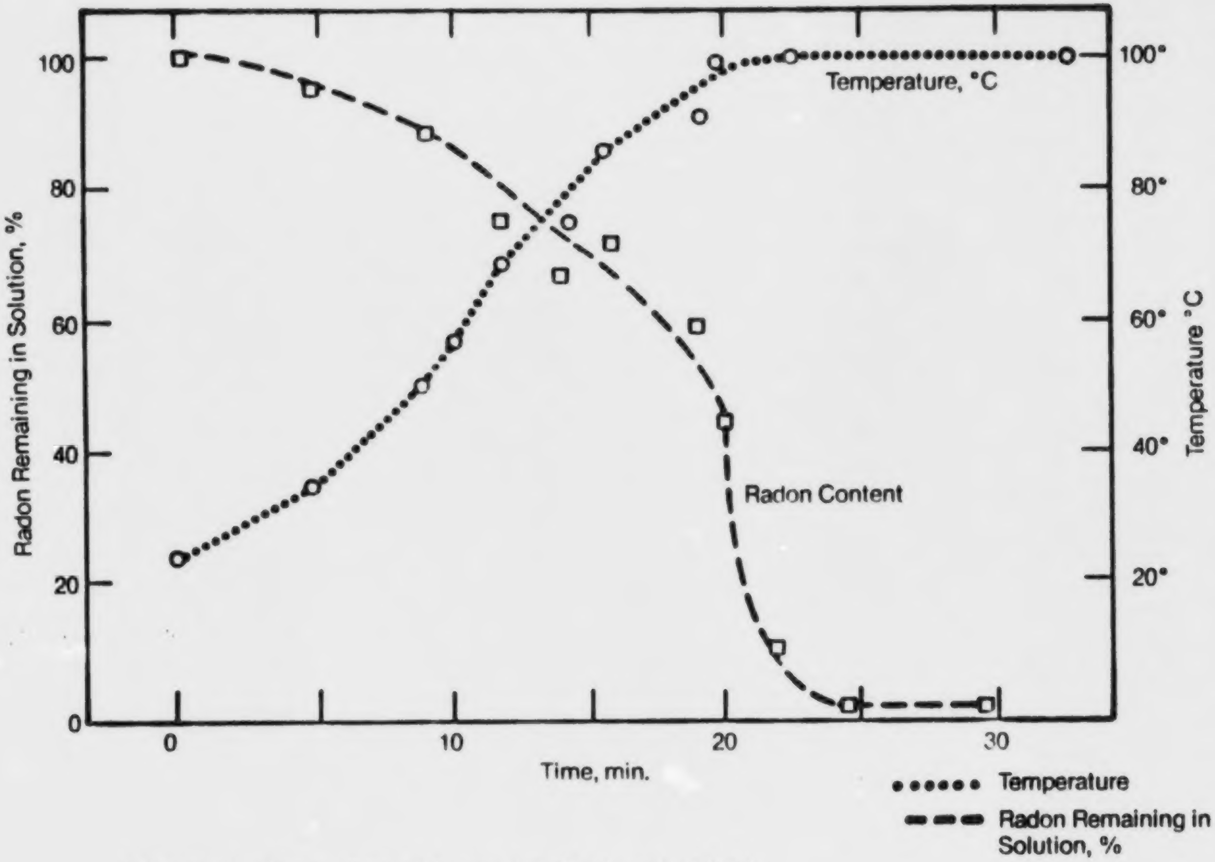
where:

C_a = concentration of radon in dwelling air (pCi/L).
C_w = concentration of radon in water used in the home (pCi/L).
F = transfer efficiency of radon from water to air.
V_w = household water use (L/hour).
λ = ventilation rate, expressed in air changes per hour (hour^{-1}).
V_h = volume of the house (L).

Note that this equation ignores radioactive decay, since the probability of a radon atom being removed from a dwelling by decay, rather than by the ventilation, is very small. Separate values of F and V_w should be used for each use of water in the home, followed by summation over all uses of water.

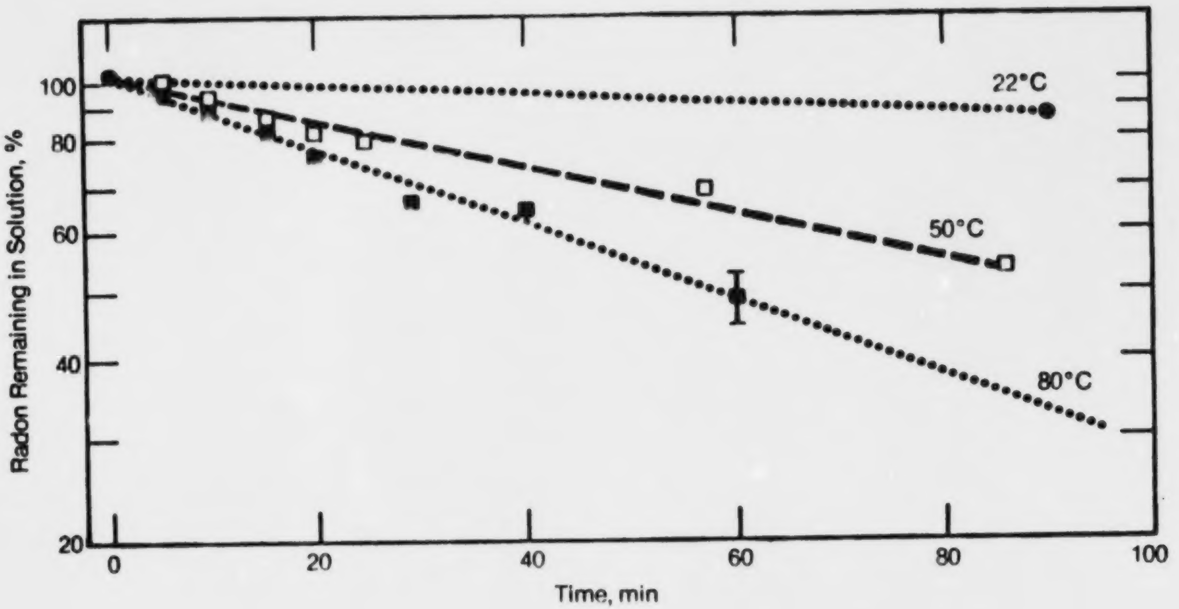
$$C_a = \frac{C_w}{(\lambda)(V_h)} \sum_i F_i V_{w,i}$$

where the summation extends over all uses of water, i.



Adapted from Gesell and Prichard 1980.

Figure IV-5 Loss of Radon as Water is Heated and Boiled



Adapted from Gesell and Prichard 1980.

Figure IV-6 Loss of Radon with Time from a 750-mL Unagitated Water Sample Held at Three Constant Temperatures

The above equation yields air concentrations that are explicit functions of the water concentration, the ventilation rate, the house volume and the specific uses of water. Each of these variables can vary drastically between homes in the United States. Daily water usage, for example, depends on the number of individuals in a household as well as on such personal characteristics as the frequency of bathing, dishwashing, laundry, etc., (see Table IV-9). As displayed in previous tables and figures, the transfer efficiency for a given water use is a function of the water temperature, which will also vary between homes. The ventilation rate depends upon the degree to which a dwelling has been sealed or insulated, primarily in response to considerations of energy efficiency. In general, the ventilation rate will increase as the home allows more access to air through cracks in the walls, flooring or ceilings or between and around windows and doors. In addition, the extent to which windows and doors are opened can greatly affect the ventilation rate.

In view of these many potential sources of variability in the parameters of the last equation, it is expected that calculations of the air concentration of radon in a dwelling arising from the use of radon-laden water would be sensitive to the parametric values assumed. For example, UNSCEAR (1977) assumed a transfer efficiency of 100%, a water use rate of 1,000 L/day, an air change rate of 1 hour^{-1} and a house

Table IV-9 Radon Released Per Day, Assuming 100 pCi/L in the Water, for Four People

Use	Daily consumption (L)	Transfer to air (%)	Radon liberated (pCi)
Showers	150	63	94,500
Tub baths	150	47	70,500
Toilet	365	30	109,500
Laundry	130	90	117,000
Dishwasher	55	90	49,500
Drinking and kitchen	30	30	9,000
Cleaning	10	90	9,000
Total	890		459,000

volume of 230,000 L. These calculations yield an estimated ratio of air concentration to water concentration (C_a/C_w) of 2.0×10^{-4}. It should be borne in mind that the transfer efficiency of 100% used by UNSCEAR is in contrast to the average of 55% determined empirically, indicating that the estimate may be incorrect by a factor of two.

Gesell and Prichard (1980) also performed theoretical calculations of C_a/C_w for several types of dwellings. They assumed a constant water-to-air transfer efficiency of 50% and a water use rate of 37.1 L/hour. Their calculations, which were specific to a small apartment, a new brick home, an old frame house and a large older structure, resulted in values for C_a/C_w of 5.1×10^{-4}, 1.1×10^{-4}, 0.5×10^{-4} and 0.1×10^{-4}. It will be noted that the upper and lower values for C_a/C_w differ by a factor of approximately 50, indicating that values of C_a/C_w may be expected to deviate greatly between different dwellings, in which the transfer efficiency probably will not remain constant. Similar calculations were performed by Kahlos and Asikainen (1980) for typical Finnish single and double family structures. Their results indicate a ratio for C_a/C_w of 1.4×10^{-4}, which is in line with the mean value of the Gesell and Prichard (1980) calculations.

The problems associated with theoretical developments of the ratio, C_a/C_w, may be circumvented by examining empirically derived values. Direct experimental data have been obtained in dwellings in which both the water concentration and the air concentration were measured concurrently. For example, Hess et al. (1978) measured the concentration of radon in groundwater and in the air of dwellings in Maine ($N = 128$). After discussing some of the sources of measurement error, they concluded that the ^{222}Rn concentration in air is typically found to be lower than that in water by a factor of 10^{-4}, at least in the room in which water use occurs. This number corresponds quite well to the mean value obtained through calculations. Their results also suggest that the ratio, C_a/C_w, may depend strongly on the radon water concentration, since their measured values ranged from 0.59×10^{-4} to 1.2×10^{-4} when C_w was greater than 1,000 pCi/L and were equal to approximately 9×10^{-4} when C_w was equal to 100 pCi/L. It is important to note that their results for C_a/C_w do not include the contribution from radon released by sources other than water.

In a later publication, Hess et al. (1979) reported the results of measurements of C_a/C_w in ten dwellings. For the higher radon water concentrations (C_w greater than 1,000 pCi/L), values of C_a/C_w ranged from 0.4×10^{-4} to 18×10^{-4}, not accounting for variability in background levels. The mean of their measurements yields an estimate for C_a/C_w of 4.8×10^{-4}, if all measurements are used, or of 1.5×10^{-4}, if a single large value of 18×10^{-4} is excluded.

In another series of measurements designed to verify theoretical calculations, Gesell and Prichard (1980) measured C_a/C_w in dwellings. Their results ranged from 0.5×10^{-4} to 2×10^{-4}, with the higher values being associated with dwellings characterized by low ventilation rates (0.25 $hour^{-1}$). Their mean value for the four measurements is 1×10^{-4}. This agrees well with the mean value of 0.75×10^{-4} measured by Hess et al. (1981) in a sampling of 18 homes in Maine. The most recent report by these authors (Hess et al. 1985) indicates a mean value of 1.3×10^{-4} for C_a/C_w in 70 homes in the United States, discounting other sources of radon. By contrast, McGregor and Gourgon (1980) obtained a mean value of only 0.17×10^{-4} for the C_a/C_w in trailers, schools and homes in Nova Scotia, Canada.

Several other authors have reported measured values of C_a/C_w in a variety of dwellings (Partridge et al. 1979, Kahlos and Asikainen 1980, Prichard et al. 1983). Their results, as well as those previously presented, may be found in Tables IV-10 and IV-11. In general, these results fall within the range of values already discussed. Of particular note is the result reported by Hess et al. in 1981. They measured air concentrations of radon continously throughout a 7-day period in the 18 homes sampled. The data display discrete spikes (increases) in the air concentration of radon shortly after the use of water in showers, dishwashers and clothes washers. The increases resulted in air concentrations that were five to ten times background levels, indicating that radon-laden water can

Table IV-10 Summary of C_a/C_w Literature Data

Source	C_a (pCi/L)	C_w (pCi/L)	C_a/C_w	Experimental conditions/basis		
UNSCEAR (1977)	0.2	1,000	1.0×10^{-4}	Series of assumptions: 4 people, water use = 1,000 L/day, 230,000 L = V_{house}, $\lambda = 1\ hr^{-1}$, f = 1.0.		
Hess et al. (1978)	0.09	100	9.0×10^{-4}	λ = unknown, actual data based on measurements of C_a in the same room as the source. Lead authors to conclude $C_a/C_w = 10^{-4}$.		
	0.3	3,000	1.0×10^{-4}			
	0.7	9,000	0.78×10^{-4}			
	4.5	60,000	0.75×10^{-4}			
	5.0	85,000	0.59×10^{-4}			
	10.0	85,000	1.2×10^{-4}			
Hess et al. (1979)	2.4±1.2	60,000	$(0.4\pm0.2)\times10^{-4}$	$\lambda = 3.0\ hr^{-1}$	24-hr radon values in these dwellings. Wrenn-Spitz-Lundum measurements.	
	2.6±0.7	1,480	$(18\pm5)\times10^{-4}$	$\lambda = 1.1\ hr^{-1}$		
	10.3±1.6	24,810	$(4\pm2)\times10^{-4}$	$\lambda = 1.0\ hr^{-1}$		
	3.9	87,430	0.45×10^{-4}	$\lambda = 2.1\ hr^{-1}$		
	3.3	32,670	1.0×10^{-4}	School λ = unknown		
Partridge et al. (1979)	0.18	10,000	0.18×10^{-4}	$\lambda = 2.0\ hr^{-1}$	V_w = 23.3 L/hr f = 0.625 $V_{house} = 4\times10^5$ L	
	0.42	10,000	0.42×10^{-4}	$\lambda = 1.0\ hr^{-1}$		
	0.78	10,000	0.78×10^{-4}	$\lambda = 0.5\ hr^{-1}$		
	1.3	10,000	1.3×10^{-4}	$\lambda = 0.25\ hr^{-1}$		
	0.92	10,000	0.92×10^{-4}	$V_{house} = 1.4\times10^5$	V_w = 23.3 L/hr f = 0.625 $\lambda = 1.0\ hr^{-1}$	
	0.40	10,000	0.40×10^{-4}	$V_{house} = 3.4\times10^5$		
	0.18	10,000	0.18×10^{-4}	$V_{house} = 6.6\times10^5$		
Gesell and Prichard (1980)	1	10,000	1.0×10^{-4}	Estimation basis, V_{house} = 200,000 L	V_{house} (L)	(hr^{-1})
	0.51	1,000	5.1×10^{-4}	Calculated	150,000	0.25
	0.11	1,000	1.1×10^{-4}		340,000	0.50
	0.05	1,000	0.5×10^{-4}		340,000	1.0
	0.01	1,000	0.1×10^{-4}		680,000	2.0
	0.35	1,750	2.0×10^{-4}	Actual measurements	175,000	0.25
	0.04	700	0.57×10^{-4}		340,000	1.0
	0.18	2,000	0.90×10^{-4}		340,000	0.5
	0.10	2,000	0.5×10^{-4}		500,000	1.0
Kahlos and Asikainen (1980) (Finland)	unknown		1.4×10^{-4}	Housewives and small children.		
	unknown		0.6×10^{-4}	Other persons.		
	unknown		0.87×10^{-4}	Population-weighted coefficients for all of Finland.		

continued -

Table IV-10 - continued

Source	C_a (pCi/L)	C_w (pCi/L)	C_a/C_w	Experimental conditions/basis
McGregor and Gourgon (1980)	0.5	158,000	0.032×10^{-4}	Nova Scotia, Canada trailers, actual measurements.
	3.2	164,000	0.2×10^{-4}	
	0.6	152,000	0.039×10^{-4}	
	2.0	158,000	0.13×10^{-4}	
	4.1	168,000	0.24×10^{-4}	
	2.5	148,000	0.17×10^{-4}	School.
	0.5		0.034×10^{-4}	
	3.4	129,000	0.26×10^{-4}	Conventional homes.
	2.2	43,000	0.51×10^{-4}	
	0.7		0.16×10^{-4}	
	1.2	98,000	0.12×10^{-4}	School.
	0.6		0.061×10^{-4}	
	19.1	370,000	0.52×10^{-4}	Conventinal homes.
	6.6		0.18×10^{-4}	
	1.5	190,000	0.079×10^{-4}	
	3.0		0.16×10^{-4}	
	3.3	314,000	0.11×10^{-4}	
	1.2		0.038×10^{-4}	
NCR (1981)	0.2	1,000	1.0×10^{-4}	General statement.
			$0.75\pm0.1\times10^{-4}$	
Hess et al. (1981)	1.9	52,000	0.37×10^{-4}	
	1.7	17,000	1.0×10^{-4}	
	3.2	27,000	1.2×10^{-4}	
	0.7	6,500	1.2×10^{-4}	
	4.5	28,000	1.6×10^{-4}	
	3.0	18,000	1.7×10^{-4}	
	<0.3	330	9.1×10^{-4}	
	<0.3	330	9.1×10^{-4}	
	<0.3	330	9.1×10^{-4}	
	1.5	22,000	0.68×10^{-4}	
	1.5	25,000	0.60×10^{-4}	
	1.0	8,000	0.13×10^{-4}	
	5.0	28,000	0.18×10^{-4}	
	<0.3	330	9.1×10^{-4}	Normalized to $\lambda = 1\ \text{hr}^{-1}$
	3.8	52,000	0.73×10^{-4}	Graphically: $(0.6\pm0.1)\times10^{-4} = C_a/C_w$
	0.85	17,000	0.50×10^{-4}	Add 25% for weak sources
	1.6	27,000	0.59×10^{-4}	$(0.75\pm0.1)\times10^{-4} = C_a/C_w$
	0.35	6,500	0.54×10^{-4}	
	2.0	28,000	0.71×10^{-4}	
	1.0	18,000	0.56×10^{-4}	

continued -

Table IV-10 - continued

Source	C_a (pCi/L)	C_w (pCi/L)	C_a/C_w	Experimental conditions/basis
Hess et al. (1982)			$(0.8\pm0.2)\times10^{-4}$	Normalized to $\lambda = 1\ hr^{-1}$, corrected radon bursts by 33% to account for radon from all water.
UNSCEAR (1982)	-	-	1.5×10^{-4}	f = 1.00 (NEA 78)
	-	-	1.0×10^{-4}	(Du 76)
	229 (avg. 32 obs.)	138,000 (avg. 20 homes)	20.6×10^{-4}	Avg. C_a/C_w for 32 rooms, situations where much water used (showers).
	78 (avg. 47 obs.)	138,000	5.64×10^{-4}	Avg. C_a/C_w for 47 rooms, situations where little water used (cooking).
	5.92 (avg. 20 obs.)	138,000	0.60×10^{-4}	Avg. C_a/C_w for 20 living rooms, situations where no water used (An 78).
Hess et al. (1983)			1.3×10^{-4}	Avg. C_a/C_w in 70 homes discounting other sources (not normalized for λ).

Table IV-11 Increment of Radon in Indoor Air Due to Radon in Water

(pCi/L in air per 10^5 pCi/L in water)

Factor	Reference	Conditions
14	Kahlos and Asikainen (1980)	Calculated value for typical Finnish single and double family structures, based on experimentally determined radon releases.
	Gesell and Prichard (1980)	Calculations based on 4 occupants, experimentally determined radon releases. Volume Air Change Rate
50		15000 L 0.25 hr^{-1}
10		34000 L 0.50 hr^{-1}
5		34000 L 1.00 hr^{-1}
4	Prichard et al. (1983)	Observation obtained by regression from 3 months integrated data from 80 houses in Maine, causality not strictly implied.
< 1	McGregor and Gourgon (1980)	Air and water grab samples in 13 structures in Halifax, Nova Scotia.

be a strong factor controlling indoor levels of radon. As expected, the duration of the increase was a decreasing function of the ventilation rate in the dwelling. The discussion of measurement uncertainties provided by the authors also applies to the other cited studies:

(1) While the C_a is measured frequently and by several techniques, the C_w appears to be based on a single grab sample.

(2) Measurements of C_a often have a lag time for response. If there is a lag time for which no correction is made, values of C_a/C_w could be in error, if the value does not remain constant with time. This problem usually is accounted for in studies.

(3) Measurements of C_a depend on humidity and require application of a previously determined correctional factor.

(4) Ventilation rates are only poorly measured in most instances.

On a final note, the National Research Council (NRC 1981) states that "a concentration of 10,000 pCi/L can raise average indoor ^{222}Rn content by about 1 nCi/m^3." This corresponds to a value for C_a/C_w of 1×10^{-4}. Unfortunately, this source provides no explanation or reference for this value. In a recent report (Cross et al. 1985), a group studying the health effects of radon in water employed values of C_a/C_w ranging from 1×10^{-4} to 2.5×10^{-4}, while a similar group on the occurrence of radon in water (Hess et al. 1985) assumed a mean value of 1×10^{-4}. From these and other results cited previously, it appears that a value of 1.0×10^{-4} for the ratio, C_a/C_w, is a good estimate of the mean value for dwellings in the United States, although wide variability among specific dwellings may be expected.

D. Other Sources of Radon in Dwellings

Other sources of radon emanation into the home are important and must be considered for comparison of their relative contribution. As discussed earlier, radon is produced in soil and rocks due to the presence of ^{226}Ra. This radon can diffuse through soil and into structures through foundation cracks, basement drains, etc., and by movement into outside air, followed by translocation into the house.

The concentrations of radon and progeny in homes as a result of soil or rock outgassing will depend upon the rate of exhalation, the ventilation rate in the home and the elevation of any specific room above the ground. The concentration in houses as a result of this soil or rock outgassing often will dominate the others (Hess et al. 1985), with subsurface emanation rates varying between 0.005 and 1.9 pCi/m^2-sec and with a mean value of approximately 0.4 pCi/m^2-sec (Becker and Lachajczyk 1984). These same authors have employed a computational model of radon build-up in a ventilated home to estimate the air concentration in a dwelling as a result of radon emanation from soil and rocks. For a house of

230,000 L, with a floor area of 92 m^2 and a ventilation rate of 1 $hour^{-1}$, they calculated an air concentration of 0.007, 0.605 and 2.74 pCi/L for emanation rates of 0.005, 0.42 and 1.9 pCi/m^2-sec, respectively. This compares well with the estimate by Hess et al. (1985) that soil radon contributes approximately 0.03 to 1.5 pCi/L in houses with normal ventilation. This contribution may be compared with the normal range of radon concentrations in homes, which typically extends from 0.3 to 2.2 pCi/L. It is apparent that radon emanation from soil and rocks contributes a substantial fraction of the radon normally found in homes.

Construction materials are also a significant source of radon in dwellings, although they provide less than that outgassed from soil. A number of recent publications have examined the extent to which building materials may contribute to indoor levels of radon (Eichholz et al. 1980a,b, Abu-Jarad and Fremlin 1983, Bruno 1981, 1983). These reports concur that radon produced from ^{226}Ra in construction materials can diffuse through the material and enter the dwelling air. The major factors controlling the extent of radon emanation into the home from such materials are (1) the ^{226}Ra content of the material, (2) the moisture content of the material, (3) the presence and nature of surface coatings, (4) the ventilation rate in the dwelling and (5) the thickness of the building material (Becker and Lachajczyk 1984). In a summary of results of measurements on radon exhalation from building materials, Bruno (1981) concluded that the contribution from such materials yields between 0.003 and 0.3 pCi/L to houses in the U.S. This source, therefore, contributes on the order of 10% of the radon brought in by soil outgassing.

The most well-known instance of construction materials yielding radon exposures in dwellings arose in Grand Junction, Colorado. Uranium mill tailings that contained elevated levels of ^{226}Ra were used extensively as fill soil around foundations of dwellings. The resulting problem led to a remedial action program to remove the tailings from those structures in which the indoor radon concentration exceeded the recommendations of the Surgeon General's report. A similar problem arose in connection with the phosphate mining area of Florida, where dwellings were built partially on phosphate rock residues, which are also high in ^{226}Ra content. Finally, it should be noted that while construction materials generally contribute significantly less radon to houses than does soil outgassing, use of gypsum wall board constructed from residues of phosphate processing or of cinder blocks made from fly ash or blast furnace slag can result in radon concentrations of over 10 pCi/L (USEPA 1979).

The last source of radon in dwellings that will be considered here is from fuel gas. Barton (1971), Johnson et al. (1973) and Gesell (1974) have each estimated radon increments from the use of natural gas as a fuel, while Gesell (1977) and the authors above have performed similar calculations for LPG (liquified petroleum gas). The contributions from these sources were low and were due primarily to the wide use of ventilators with such sources. The mean contribution from these sources is estimated to be approximately 0.05 to 0.15 pCi/L in the U.S.

A summary of some measured values of background levels of radon in homes in the U.S. may be found in Tables IV-12 and VI-13. These measurements include radon from water sources, although, to the extent possible, they reflect radon during times of low water use, so that the water component is minimized. These results indicate a geometric mean radon concentration in the region of 1 to 2 pCi/L, which is in agreement with the above discussion and with a series of measurements performed by a monitoring group at Oak Ridge National Laboratory (Myrick et al. 1981). Canadian values have been reported to range from 0.14 pCi/L in Vancouver to 0.88 pCi/L in St. Lawrence, Newfoundland, with log normal distributions characterized by geometric standard deviations of between 3 and 7 (McGregor and Gourgon 1980). If a mean value of 1 pCi/L is assumed for the U.S. and an equilibrium factor of 0.5 is employed, the average radon progeny concentration may be shown to be equal to 5×10^{-3} WL. Approximately 50% to 75% of this radon will arise from soil and rock outgassing (both directly to the indoor space and indirectly into outdoor air), with another 10% to 15% being contributed by building materials and

Table IV-12 Indoor Radon Concentrations (pCi/L) in "Background" U.S. Dwellings

Site	Number of dwel-ings	Geom. mean	Geom. S.D.	Eql. ratio	Comments	Reference
New York and New Jersey						
Basements	18	1.7	2.0	0.48	Ordinary houses	Gesell & Prichard (1980)
1st Floor	18	0.83	2.0	0.49		
2nd Floor	9	0.77	1.8	0.45		
Central Maine						
Basements	77	2.46	2.4		Ordinary houses	Prichard et al. (1983a)
1st Floor	82	1.40	2.4			
Bedroom (2nd)	81	1.12	2.4			
Bathroom	81	1.62	2.4			
Outside	67	0.46	2.4			
Houston, Texas						
Bedroom	103	0.39	2.5		Houses, apts.	Prichard et al. (1983)
Bathroom	103	0.56	2.5			
Outdoors	81	0.22	2.6			
Eastern Pennsylvania						Becker 1981 (secondary source)
Living area	36					
Summer		1.22	4.6			
Winter		4.40	3.7			
Basement	36					
Summer		3.40	6.4			
Winter		5.90	4.0			

Table IV-13 Radon Levels Measured in Air in Houses (from Becker 1981)

	Average pCi/L	Maximum pCi/L	Number of houses
Maine	3.0	63.0	85
New York			
Albany area	3.1	26.0	21
New York City Area	1.0	5.9	21
California			
San Francisco	0.4	1.1	28
Pennsylvania			
Eastern	10		36
Maryland	3.7	27.0	56

natural gas. The remainder (10% to 40%) presumably arises from the radon released as a result of water use, with the lower bound being most likely.

V. HEALTH EFFECTS IN ANIMALS

A. Introduction[a]

Animal studies have been conducted for several decades to identify the nature and levels of uranium mine-air contaminants that were responsible for producing the lung cancers observed among uranium-mining populations. Many of the initial studies were concerned with early effects or short-term pathological changes (Jansen and Schultzer 1926, Read and Mottram 1939, Jackson 1940). Exposures were based primarily on radon gas concentrations, giving little or no information on the radon-progeny concentrations, which subsequently were shown to contribute the greatest radiation dose to the lung. The early studies (Huech 1939, Rajewsky et al. 1942a, 1942b, Kushneva 1959), in which lung tumors were produced, were methodologically or statistically inadequate to show an unequivocal association of lung tumors with exposure to radon and/or radon progeny.

Beginning in the 1950s, a growing concern emerged that the increased incidence of respiratory cancer observed in the European uranium-mining population would also be found in the U.S. mining population (Seven State Mining Conference 1955, Wagoner et al. 1964). Systematic studies were subsequently begun in the U.S. to identify the agents responsible for the increased incidence of lung cancer in miners and to develop exposure-response relationships in animals. Investigators at the University of Rochester began to focus attention on the biological and physical behavior of radon progeny as well as their contribution to the radiation dose to the respiratory tract (Bale 1951, Harris 1954, Morken 1955). Shapiro (1954) exposed rats and dogs to several levels of radon alone and in the presence of radon progeny attached to room-dust aerosols. He also showed that the degree of attachment of radon progeny to carrier dust particles was a primary factor in influencing the -radiation dose to the airway epithelium. He demonstrated that this dose was due primarily (>95%) to the short-lived radon progeny, rather than to the parent radon.

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[a] This section is reproduced in its entirety from a report by Cross et al. (1985).

In 1953, Cohn et al. (1953) reported the relative levels of radioactivity found in the nasal passages, in the trachea and major bronchi, and in the other portions of rat lungs after exposure to radon and/or radon progeny. The respiratory tracts of animals that inhaled radon plus its progeny contained 125 times more activity than those of animals that inhaled radon alone, indicating that the effects noted in animals breathing radon plus progeny probably were due to the progeny and not to the radon itself.

Beginning in the mid 1950s, Morken initiated a pioneering series of experiments (Morken and Scott 1966, Morken 1973a, 1973b) to evaluate the biological effects of inhaled radon and radon progeny in mice; later experiments used rats as well as beagle dogs. The essentially negative biological results of these studies (due primarily, we believe, to inadequate follow-up times in the experiments) suggested that -radiation is inefficient in producing tumors in the respiratory system. The only apparently permanent late changes occurred in the alveolar and respiratory bronchiolar regions of the lung for a wide range of exposure levels and for observation times to 3 years in the dog and 1 and 2 years, respectively, in the rat and mouse. Furthermore, injury in the bronchial tissue was quickly repaired after irradiation ceased. The carrier aerosols used in these experiments were more typical of environmental aerosols than of those found in the mines.

In the late 1960s and early 1970s, other studies in France and the U.S. were initiated, which later proved successful in producing lung tumors from inhaled radon progeny. The French exposed rats either to radon progeny alone or in combination with stable cerium, uranium-ore dust or cigarette smoke to produce tumors in the lung (Perraud et al. 1970, Chameaud et al. 1974, 1980). The later U.S. studies were designed to systematically determine the pathogenic role of radon progeny alone, or in various combinations with uranium-ore dust, diesel-engine exhaust and cigarette smoke. These studies involved lifespan exposures of beagle dogs and Syrian Golden hamsters and chronic exposures of rats (Cross et al. 1978, 1982).

A discussion of the biological effects in animals of inhaled radon and radon progeny is included in the recent ICRP Publication 31 (1980). In general, the lung cancers in rats were noted to be about half bronchogenic and half bronchiol alveolar in origin, in contrast to the nearly exclusive bronchogenic origin of human lung cancers. Extrapulmonary lesions were not a significant finding in the radon inhalation studies. An even more detailed presentation of the animal studies is presented in NCRP Report No. 78 (1984a).

B. Discussion and Summary

The animal studies have provided considerable data confirming the human epidemiology studies:

1. In rats, primarily, tumor production per WLM at very high exposures was lower than at moderate exposures (Cross et al. 1982, Chameaud et al. 1980).

2. In both the human and animal studies, tumor prodution appeared to increase with the decrease from high exposure rate (Chameaud 1980, Cross et al. 1982); the influence of exposure rate is unknown at current occupational and environmental exposure levels.
3. In rats, tobacco smoke was found to be co-carcinogenic with radon progeny when exposure to the smoke followed completion of exposure to the progeny (Chameaud et al.

1980). This effect was not observed, however, when smoking preceded the radon progeny exposure by a short period of time (Chameaud et al. 1981). Such disparities may partially explain discrepancies in interpreting epidemiological data (since initial exposure to smoking may stimulate mucus flow, while prolonged exposure could thicken and slow the blanket.

4. Emphysema and fibrosis have been attributed to radon progeny exposure in animals--hamsters, rats and dogs (Stuart et al. 1978, Cross et al. 1978). Simultaneous exposure to ore dust or diesel exhaust increased the incidence of these lesions but did not appear to increase the number of tumors produced by exposure to radon progeny (Cross et al. 1978, 1982, Chameaud et al. 1981).

5. For equivalent, cumulative, radon progeny exposure, the older the animal at the start of exposure, the shorter the latent period (Chameaud et al. 1981).

6. The predictions of the various dosimetric models appear to be borne out in the various species. The tumors induced in experiments with animals are commonly more distal than those in humans. Desrosiers' (1978) modeling of Syrian Golden hamster lungs showed that peripheral basal and Clara cells may receive

doses greater than, or approximately equal to, those received by basal cells in the central airways.

7. Lifetime risk coefficients are similar in both animals and humans. The coefficients based on rat data appear to range between 1 and 4 x 10^{-4}/WLM for all tumors (benign and malignant) at cumulative exposures less than 5000 WLM (Chameaud et al. 1981, Cross unpublished data). At exposures considerably lower than where lifespan is significantly shortened (<500 WLM), the lifetime risk coefficient appears to be about 2 x 10^{-4} per WLM for malignancies and ranges between 2 and 4 x 10^{-4} for all tumors. Data are as yet insufficient to determine a value for exposure below 100 WLM.

Lung cancer is not the only disorder arising in the lungs of animals exposed to radon and its progeny, although it certainly has received the most attention due to its apparent dominance in risk considerations. For example, the studies on mice and dogs at the University of Rochester (Morken and Scott 1966) indicate that destructive, hyperplastic and metaplastic lesions appeared in the lungs in much greater numbers than did tumors. An interesting point here is that lesions in the bronchial tree often were quite quickly repaired following termination of exposure. Many of the other animal studies demonstrated the production of pulmonary fibrosis, emphysema and nasopharyngeal hyperplasia, but each of these studies included exposure to uranium ore dust, presumably the cause of the destructive effects.

VI. HEALTH EFFECTS IN HUMANS

This section describes the human health effects linked to, or implied by, exposure to airborne radon in the home. It is assumed that these exposures arise from the emanation of radon from the water into the air of a dwelling. There is also a brief discussion of the risks from ingested radon and immersion exposures. The section begins with a simple discussion suggesting causality between exposure to radon progeny and lung cancer. Epidemiological data from both humans and other animals are then examined with the intent of developing an appropriate risk factor (risk per WLM) for human exposure to environmental levels of radon progeny in air. This factor then results in a predictive model for lung cancer induction, as outlined in the report by Cross et al. (1985).

A. Causal Links Between Radiation and Lung Cancer

A presupposition implicit in this report is that low levels of radiation (in particular, alpha radiation) are capable of inducing a sufficient number of cells to transform to the cancerous state. The validity of such an assumption is taken for granted in most calculations of the effect of the radon decay chain on the human body. While a complete defense of such a position would be beyond the scope of this report, it was felt that some attention should be directed toward the strength of causality in radiation-induced lung cancer in general and in the radon decay chain in particular. In addition, there is a need to specify the critical cells at risk. This section begins with a short discussion of the induction of lung carcinomas by inhaled and injected radionuclides.

Current belief holds that all human cancers are inducible by radiation. Kennedy and Little (1978) have recently published a review of the available data linking exposure to radiation and the development of lung cancers. Their document essentially completes the reports of Cember (1964), which reviewed data available through 1964, and Bair (1970). As in all studies of causality, the evidence is divided into two groups: epidemiological and experimental. A third category, theoretical, might be added by noting that no empirical evidence can be used to prove causation without the development of a theory describing some underlying mechanism of action (Eddington 1958). Kennedy and Little reviewed the first two categories and concluded that "lung cancer has now been induced by various kinds of radiation in many species of animals using several methods of exposure" (Kennedy and Little 1978). No attempt will be made to review the data presented in that document, other than to inform the reader that cancers have been induced by gamma, beta and alpha irradiation, with beta and gamma radiation being less efficient per unit dose than the short-range alpha particles. Additional reviews may be found in the UNSCEAR (1977) and BEIR reports (NAS 1980) and in the reports by the NCRP (1975) and the National Academy of Sciences (NAS 1976). Each of these authoritative bodies conclude that available evidence suggests a direct causal link between radiation delivered dose and pertinent cancers(stomach, lung, liver, and kidneys), at least at high doses. In their recent report, the NCRP (1984a) conclude that the link between lung cancer and exposure to radon progeny at high levels is strong.

The current philosophy of epidemiology holds that it cannot be used to "prove" causality in any real sense (Eddington 1958). One is able, however, to test the strength of an association between exposures and incidence. This strength is typically measured through five characteristics (Lowrance 1976):

(1) The consistency of the association.
(2) The strength of the association.
(3) The specificity of the association.
(4) The temporal relationship of the association.
(5) The coherence of the association.

These categories have been utilized by authors to examine data pertinent to the induction of lung cancers by irradiation. It is possible to divide the data into two groups: those which are directly related to the radon problem and those which provide peripheral evidence. The A-bomb data and three different groups of patients exposed to radiation through medical diagnosis fall in the latter category. These may be used to demonstrate an association between radiation exposure in general and the induction of lung cancer. The Atomic Bomb Casualty Commission (ABCC) Life Span Study (Jablon and Kato 1972, Beebe 1978) report has utilized the A-bomb data to demonstrate an increase in lung cancer incidence which appeared to be directly associated with exposures received during or immediately after the explosion of the bomb. The BEIR report (NAS 1980), however, states that difficulties exist in interpreting these data, since the observed cancer rate in the control group was "about 50% higher than that expected for the Japanese at large" and since there was an uncertainty as to the contribution from neutron irradiation. Other groups which have exhibited increased risks of lung cancer include ankylosing spondylitis patients (Court Brown and Doll 1965), tuberculosis patients given fluoroscopy diagnosis (Steinitz 1965), x-ray technicians (Miller and Jablon 1970) and patients receiving injections of thorium dioxide as a contrast agent (Horta and daMotta 1967). Once again, the association is significant, although competing factors may have been present. These same conclusions have been reached by the BEIR committee in their most recent report (NAS 1980).

Such studies as the ones cited above have been utilized to develop a risk/dose conversion factor for lung cancer. A risk factor for the induction of lung cancer by the radon chain would then require a knowledge of the radiation dose and the appropriate modifying factors. These modifying factors include differences in the relative distribution of dose between the various subpopulations in the lung. The dose itself is difficult to calculate, but the modifying factors are even more uncertain. Therefore, it is desirable to explore any information concerning irradiation under circumstances similar to those encountered during inhalation of the radon chain in dwellings. As was discussed earlier, this information is partially available through the long history of exposure of miners to the radon decay chain. The significance of the current research hinges on the ability to demonstrate a direct causative link between exposure to radon and the induction of lung carcinomas.

All of the studies cited earlier [which consist of the Schneeberg (Sevc and Placek 1972), Joachimsthal (Pierquin et. al 1965), Canadian fluorspar (de Villiers and Windish 1964, Parsons 1964), Colorado plateau (Archer 1972, 1976), Czechoslovakian (Sevc et al. 1976) and Swedish (Axelson and Sundell 1978) miners] have found a direct link between the amount of exposure to radon and its progeny and the incidence of lung cancers in the population. This link has satisfied the five requirements for causality postulated earlier, unlike the relatively poor links discerned with other forms of cancer. Complete reviews of this information may be found in the articles by Archer et al. (1980, 1973), Wagoner et al. (1964), Donaldson (1969) and Sevc, et al. (1976), as well as in the recent paper by Whittenmore (1980) and in NCRP report 78 (1984a). The question arises, however, as to whether there might be another agent in the mines which is the true causal factor, with radiation acting only as a co-factor. A number of agents have been suggested, including silica, compounds of cobalt and nickel, bismuth and arsenic. Of these, only arsenic has shown any significant correlation with lung cancer induction, although exposure to silica has produced numerous instances of silicosis. Weller (1965) and Heuper (1942) have reviewed the nature of this correlation, and the general opinion is that only radiation shows a consistently significant correlation among all study populations. The possibility of the synergistic or cocarcinogenic effects of any of the above agents has never been ruled out, however. Much more work is required in this area before any firm conclusion may be drawn. In the interim, the health physics community is acting on the assumption that inhalation of radon and its radioactive progeny leads to an increased risk of lung cancer and this assumption has been supported in the recent NCRP report (1984a).

Lung cancer actually encompasses a wide range of histologic types. Some care must be taken to ensure that the dose from the radon in mines and dwellings is delivered to the same critical cells at risk assumed here to be the basal cells of the bronchial epithelium. Three bronchogenic carcinomas are found in humans and are typically designated as squamous, glandular and undifferentiated. The squamous-celled tumors usually predominate in non-irridiated humans (Weller 1965, NAS 1976). In the mining groups, Wagoner et al. (1965) and Kotin et al. (1966) found that more than 60% of the histologic types fall into the category of small-cell, undifferentiated (oat cell) tumors, although the later work of Saccomanno et al. (1982) suggest that the prevalence of such tumors may be less, particularly as the follow up time is increased.

B. Human Epidemiological Data

As outlined above, data from a range of both occupational and non-occupational exposures have strongly suggested that radiation can cause both lung cancer and cancers of other organs. These studies could be used to develop a general understanding of the risk factor for lung cancer induction, with this factor being expressed in units of risk per rem. It would then be necessary to calculate the absorbed dose (in rads) delivered to the critical cells by inhaled radon progeny, multiply by an appropriate quality factor and apply the risk factor from the pooled data on lung exposures from a wide range of sources (as would be done in using the ICRP 26 system of calculation).

Unfortunately, quality factors are difficult to obtain in practice. As mentioned previously, the ICRP currently recommends a quality factor of 20 for alphas. Figure VI-1 displays the functional relationship between quality factors (QF) and Linear Energy Transfer (LET) for radiations, as reported and recommended by the ICRP (1977). Table VI-1 is a listing of discrete values of the QF for specific radiations.

A number of authors have recently attempted to develop an empirical value for the QF for the induction of lung cancer by the radon decay chain. The NAS reviewed animal data and compared lung cancer incidences following external gamma irradiation and internally deposited alpha- and beta-gamma-emitters. They concluded: "While alpha irradiation was generally more effective than uniform irradiation and beta irradiation, for all dose levels and all animal species the mean differences were less than 10, the value usually taken as the quality factor for alpha irradiation." Walsh (1976) reached a similar conclusion and utilized available epidemiological studies to develop a rem/rad conversion factor of approximately four (Walsh 1979). This value agrees well with that developed in the Interagency Uranium Mining Group (Archer et al. 1971). It should be borne in mind, however, that such approaches require an accurate specification of absorbed dose, a quantity which is difficult, at best, to obtain for internal emitters in the lung. Bernhardt (1973) found that the QF ranged from 1 to 20 in the literature, with a mean at 3. Both Gesell (1973) and the Federal Radiation Council (FRC) (1967) have published similar values. In summary, it would appear that use of a QF of 10 or 20 will yield a conservative estimate of dose equivalent, with a reasonable lower limit being determined through the use of a QF of 3.3.

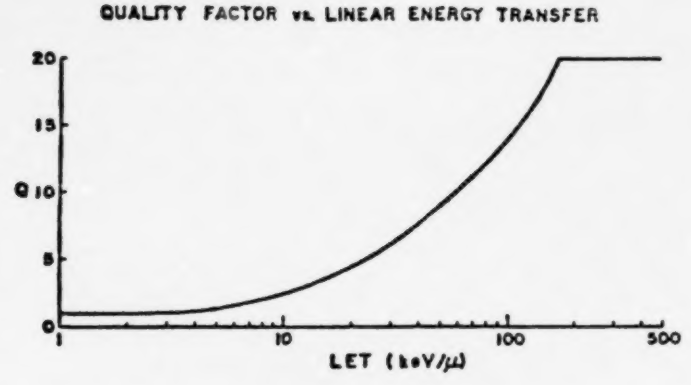


Figure VI-1 Plot of the Quality Factor Versus LET

Table VI-1 Discrete Values for the Quality Factor, Q, as a Function of the LET as Recommended by the ICRU

L in water (keV/micrometer)	Q
3.5 and less	1
7.0	2
23.0	5
53.0	10
175.0 and above	20

The problems associated with specifying QFs may be alleviated, however, by the fact that risk factors for radon progeny exposures can be obtained without reference to a computed dose equivalent. Most approaches to the development of a risk factor for environmental exposures to radon progeny, therefore, use risk factors obtained directly from exposures in uranium mines, expressed in units of risk per WLM, followed by corrections for the appropriate value for the conversion factor from WLM to rads. These corrections occur due to differences in attached fraction, breathing rate, equilibrium factors, etc. This approach was adopted recently by the NCRP (1984a).

Before detailing the results of these human epidemiological studies in mining groups, however, a few caveats should be noted. Accurate risk estimates can be obtained only if the correct exposures in mines can be assigned to workers and if the follow-up periods of study are sufficiently long. Unfortunately, neither of these criteria holdsup well in the available mining data. Exposure estimates were usually crude, being based on extrapolation of measurements from a few mines. In addition, follow-up periods were typically shorter than desired, rarely extending to the end of life for the exposed cohorts. As a result, risks factors developed from these studies have inherent limitations. The latter limitation may be overcome by continued follow-up of the cohorts, but the former is unlikely to be relieved due to the paucity of historical data and the problems associated with reconstruction of the mining atmospheres. Nevertheless, it is possible to estimate a mean risk factor as derived from direct human epidemiological studies, bearing in mind the associated problems.

As described earlier, human epidemiologic data obtained from groups of underground metal-ore miners in Czechoslovakia, Canada, the United States, Sweden, Britain and Newfoundland are currently available. These data have been summarized and reviewed by many organizations and authors (NIOSH 1971, NAS 1972, NAS 1980, Jorgenson 1972, Snihs 1973, Renard 1974, deVilliers and Windish 1964, Wright and Couves 1977, Evans et al. 1981, NCRP 1984a). Table VI-2 displays a summary of results as published by Walsh (1979).

From the published data, absolute risk values of between 1.5 and 50 cancers/WLM/year/10^6 persons may be noted for occupational exposures. A best estimate of the mean risk coefficient of 10 x 10^{-6} person/ year/WLM was published by Cross et al. (1985) and adopted by the NCRP (1984a). This value was employed in the lung cancer prediction model of Harley and Pasternack (1981) for environmental exposures to radon progeny. Walsh, on the other hand, employed relative risk methods to obtain a mean risk estimate of 1.4% increase in lung cancer incidence/WLM for adults. This agrees well with the results of Sevc et al. (1976), Archer et al. (1980), Hewitt et al. (1976) and others. It is interesting to note that if one employs the dose-to-dose equivalent conversion factor given by Walsh (1979), the risk per rem from exposure to ^{222}Rn progeny is in agreement with data obtained from exposure to x- and gamma-rays.

C. Risks to Other Organs

Lung cancer risks from inhalation of radon progeny in dwellings may be estimated directly from experience with the uranium miners. Unfortunately, the risks to other organs of the body have not been developed as fully, and results obtained from situations dissimilar to inhalation or ingestion of radon must be employed. Most of these studies involved exposure to low LET radiations, necessitating the use of an alpha QF if the low LET results are to be extended to the exposures of interest in this report. This subsection of the report details the risks of cancer imposed by doses delivered to internal organs as a result of ingestion of radon-laden water. It should be kept in mind that lung cancer risks from radon progeny produced by emanation of radon into dwelling air will dominate risk calculations, so that the present discussion is included only for completeness.

The risk factor for stomach cancer (and the G.I. tract in general) is not very well developed due to the apparently low radiosensitivity of the organ. This low radiosensitivity

Table VI-2 Excess Relative Risk (in Percent) of Radiogenic Lung Cancer in Groups Exposed to Alpha Particles, x-rays, gamma-rays and Neutrons

		Percent Increase in Excess Relative Risk		
Exposed group	Type of radiation	per WLM	per rad	per rem
Uranium miners				
U.S. (white)		0.9		
U.S. (Indian) WLM > 300		0.4		
U.S. (15 years after start), WLM < 500		0.9		
Canada		0.9		
Czechoslovakia		1.2		
Fluorspar miners		3.0		
Metal miners		0.5		
Swedish metal miners		3.2		
Thorotrast (Portugal)			1.0	
Bomb survivors			0.3	0.3
			1.0	0.2
Spondylitics	x-rays		0.2	0.2

results in poor estimates of the risk when epidemiological studies are conducted. There is evidence that radiation does induce some stomach cancers at moderate doses (ICRP 1977), but the ICRP estimates that the risk factor (risk/rem) is low. In Publication 26, the ICRP suggests that the lifetime risk of stomach cancer probably does not exceed 10^{-5}/rem.

The lifetime risk factor for whole body irradiation from uniformly distributed sources has been estimated by the ICRP to lie in the region of 10^{-4}/rem (ICRP 1977). This factor would apply to the radon which travels from the stomach to the internal organs, if uniform organ distribution is to be assumed. In addition, the ICRP states that tissues such as muscle and adipose tissues (fat) display essentially no radiosensitivity for tumor induction even at moderate doses. As a result, the component of dose equivalent delivered to adipose tissue following ingestion of radon should be of little concern for regulatory purposes. Since, as discussed earlier, some 50% to 75% of the alpha decays that occur in organs other than the stomach or G.I. tract take place in the adipose tissue, risk estimates based on the calculation of dose averaged over the total soft tissue mass dose and application of the whole body risk factor of 10^{-4}/rem should result in significant over-estimation of the risks from radon translocated from the stomach to the other internal organs.

By way of comparison, Cross et al. (1985) employed the above risk coefficients and conservative estimates of stomach and whole body doses to calculate lifetime risks from use of radon-laden water in homes. Their estimate of the lifetime risk from stomach cancer is an order of magnitude lower than that of lung cancer, while the lifetime risk from irradiation of the other internal organs is lower than that of lung cancer by a factor of 30 to 75. Since the estimate of risk of stomach cancer is very likely to be a significant overestimate, it is clear that the risk of lung cancer following inhalation will be the limiting factor in any consideration of the radiological significance of the use of radon-laden water in dwellings.

VII. MECHANISM OF RADIOTOXICITY

A. Introduction

Section VI of the present report outlined the evidence linking exposures to radon progeny and the induction of lung cancer. The conceptual basis for calculating lung cancer risk following exposure rests upon computation of absorbed dose to critical cells within the lung mass. While there currently exists only an incomplete understanding of the mechanism by which radiation exerts an influence in the induction of cancer, the concensus of the scientific community is that cancer induction probabilites by radiation are related functionally to the absorbed dose equivalent, which is a measure of the energy deposited by radiation in the critical cells, modified by a quality factor to account for the differences in effectiveness of energy deposited with differing microscopic distributions (such as differing LET). Since the calculation of dose equivalent is common to all recommendations by scientific bodies concerned with radiation bioeffects(NCRP, ICRP, etc.), it will be assumed here that no further defense is needed for proceeding on the assumption that the mechanism of radiotoxicity involves the deposition of energy in pertinent cellular sub-populations of the lung (basal or K cells of the bronchial epithelium). Instead, attention is directed towards radiobiological and physical considerations which might affect the applicability of existing data on uranium miners in estimating the radiobiological effects likely to be found at the much lower levels of radon progeny exposures in homes in the United States.

The preceding, necessarily brief, discussion was intended to suggest several observations central to this report. These are that:

1. There is a strong association between incremental radiation exposures and increased incidence of a variety of human cancers.

2. This association is extended to lung cancer, with alpha particles showing a propensity for the induction of this disorder.

3. Inhalation of the radon decay chain has been shown to be associated with the induction of lung cancers in a statistically significant manner.

4. The cancers arising from exposure to the radon decay chain are primarily in the basal cells of the third to fifth generation of the lung.

A fifth observation, although not specifically discussed, states that the incidence of lung cancers among the various study populations is not adequately accounted for by the beta and gamma doses delivered by exposure to radon progeny. This strengthens the following statement, which will act as a basic presupposition in this report:

> The induction of basal cell cancers in the lungs of humans exposed to an atmosphere containing the ^{222}Rn decay chain arises principally from the deposition of energy by alpha particles within the basal cell nuclei.

Strictly speaking, the uranium and non-uranium miner data can only be employed to estimate health effects at doses in the same range as those observed. Doses from the use of radon-laden water in homes (in the concentration noted here), however, are much lower, falling below the range of exposures typically associated with occupational limits. The question arises as to whether there is evidence to support cancer induction at these low levels and how best to utilize existing data in extrapolations to the required doses. The following discussion is directed toward a partial answer to these questions.

B. Extrapolation of Miner Data to Low Doses

Since the data from miner exposure revolve around radon progeny exposures at doses that are significantly higher than those encountered in homes, the question arises as to whether risk factors obtained at high doses are applicable to the low doses typical of environmental exposures of below 10 WLM. This question may be further subdivided into two parts: whether a threshold dose exists and, if not, whether the true shape of the pertinent dose-response curve is linear.

As to the issue of a threshold, available data neither support nor rule out a threshold for lung cancer induction. Stranden (1980) and Evans (1967) interpreted the mortality data for the mining popoulation as displaying cancer rates at low doses that are not significantly different from "background" levels. Both Snihs (1973) and Archer et al. (1979), however, concluded that no threshold can be seen in data for groups with exposures as low as 15 to 30 WLM. Hewitt (1976) analyzed the data from uranium miners in Canada and stated that any threshold must be below 60 WLM. These reports suggest that no threshold has been demonstrated and that accurate and reliable estimates of risk at low doses are not likely to be determined empirically from the miner data. This position was adopted recently by the NCRP (1984a) in their review of uranium miner data.

The scientific community has, in the past, employed linear extrapolations in extending high-dose data to the low-dose region. Justifications of this procedure have been founded principally on considerations of the scarcity of data and on a desire that estima'es be conservative. In the case of low LET radiations, this practice is being called into question (see, for example, the controversy over the new BEIR III report). For high LET particles, however, it would appear that such an extrapolation may be well founded, with exposures perhaps becoming even more effective (per WLM) at lower levels of exposure (Archer 1978, NAS 1980).

A number of authors have performed studies on the relation between the cumulative exposure of the miners and the increased incidence of lung cancer. Archer and Lundin (1967), Archer et al. (1973) and the NCRP (1984a) have utilized data from 18 mining groups to develop dose-response curves for the alpha-emitters of the radon decay chain. These results indicate a roughly linear plot up to a certain dose, followed by a leveling of the curve, presumably due to excessive cell killing. Little and Williams (1977) have reviewed the epidemiological data for the miner groups and concur with the linear extrapolation of Archer et al. Complications in each of these studies arise from the finite period of study. Since the latent period may be a function of absorbed dose (Lundin 1971), it is possible that the low-dose groups may not, as yet, have reached their maximum incidence of cancer. In addition, the latent period varies between (and within) species, indicating the possible need for elucidating host factors in the expression of radiation-induced lung cancer. Of course, if the general population were genetically "similar" to the miners, this problem would not arise.

Additional groups which have been studied for the shape of the dose-response curve in lung cancer have included the atomic bomb survivors, patients treated with x-rays for ankylosing spondylitis, patients receiving injections of thorium dioxide and former tuberculosis patients who underwent fluoroscopic examinations. In each instance, the dose-response curves have been approximated by a linear function for the high LET radiations, although the x-ray data on genetically homogenous groups more closely approximate a sigmoid shape. As a result, it would appear that, at least for high LET radiation (a group which includes alpha particles), there is no apparent dose threshold at low doses, and the general shape of the linear curve may be representative of the true curve underlying the data.

Most of the experimental studies, such as those of Han and Elkind, (1979), Borek et al. (1966, 1967, 1973, 1977) and Terzaghi and Little (1975, 1976a, 1976b), utilized low-energy x-rays. Han and Elkind have performed some studies on transformation frequencies in C3H/10 $T_{1/2}$ cells over a range of neutron doses. Results indicate a linear plot with a plateau at 2,000 to 3,000 rad (larger than the average dose to the basal cells of the uranium miners). Borek (1976) irradiated hamster embryo cells and plotted the fraction of transformed clones versus doses over a wide range of doses. The resulting graph indicates that a linear (plus cell-killing) model may be realistic for high LET radiation in the low-dose region. As in most studies of *in vitro* systems, the data for x-ray irradiation indicate a curve which falls between sigmoidal and linear curves.

The great majority of experimental data relies on studies of *in vitro* systems. While the *in vitro* systems probably offer the greatest hope for understanding the causal chain of events in carcinogenesis, studies of *in vivo* systems will continue to be viewed as being the most realistic for estimating effects on the human system. What do such studies tell us about the shape of the dose-response curves for high LET radiations? In addition, what is the effect of dose rate on the carcinogenic potential? Neary et al. (1963) studied the yield of chromosome aberrations in microspores of *Tradescantia paludosa* and concluded that the response for gamma-rays is a function of the square of the dose, while that for neutrons is linear. In addition, they have presented evidence that the gamma-induced yield is dependent on dose rate, but that the yield for high LET neutrons is not. These conclusions are substantiated by a number of studies on the frequency of chromosome aberrations in the lymphocytes of atomic bomb survivors (Awa 1975, although recent revisions in dose estimates may change this result) and by the frequency of pink mutation events in stamen hairs of *Tradescantia* clones (Sparrow et al. 1972). Similar results were obtained in the study of mouse spermatogonia (Russsell 1969), and oocytes (Russell 1965, 1969). Each of these studies indicates a linear relationship for dose-response for irradiation by high LET radiations and a linear-quadratic relation for low LET x-rays and gamma-rays. Studies by Upton et al. (1966, 1967, 1968), Ullrich and Storer (1979), Yuhas (1974) and others show a dependence of gamma-ray-induced mutations on dose rate. This dependence is believed to be eliminated (Upton et al. 1967, Upton 1968) when the radiation consists of high LET neutrons. The reader should recognize that these results lead to the deductive consequence that the relative biological effectiveness (RBE) of high LET radiations should increase with decreasing dose (since the efficiency of gamma radiation in inducing cancer decreases more rapidly with dose than does that of high LET radiation), a hypothesis supported by the work of a number of authors (Hall et al. 1975, Bateman et al. 1972) utilizing Sprague-Dawley rats. On the other hand, Ullrich et al. (1977) have determined dose-response curves for carcinogenesis in Balb/c mice and found a marked dependence on dose rate for neutrons. For malignant lung carcinomas, they found that decreasing the dose rate for low doses decreased the incidence. At high doses, however, this trend was reversed. As a result, one should be careful when stating that no dose rate effect has been observed for high LET radiations, particularly when dealing with mammary and lung adenocarcinomas and ovarian tumors. Due to the complexity of host-mediated hormonal and immunological reactions and the interactions of the repair system, it is certainly possible that no general statement may be made about the effect of dose rate on the response of all cell lines and organs. This introduces some uncertainty in attempting to extrapolate existing occupational data to the carcinomas induced by environmental (non-occupational) radon progeny in the lung. The consensus of opinion, however, is that dose rate effects decrease significantly for high LET radiation in organs which are not subsequently subjected to complex host regulation.

Existing data concerning *in vivo* irradiation of the lung from a variety of deposited radionuclides support this conclusion. Chameaud (1976), Filipy (1976), Morken (1974) have each presented evidence that protraction of the dose from inhaled ^{222}Rn and its progeny increases the incidence per rad. Similar studies using beta-emitting radionuclides and the alpha-emitter ^{210}Po have concurred with these results (Lisco and Finkel 1949, Cember 1964, Little et al. 1975). To indicate the danger of drawing general conclusions, however, it is of interest to note that Laskin et al. (1964) did not find a dose rate effect for ^{32}P and ^{106}Ru. The reader should also bear in mind the results of Ullrich et al. (1977), which were cited earlier. Most of the evidence appears to indicate that linear extrapolation will not over-estimate the incidence of lung cancer, when high LET radiations are the primary determinant of dose, and that there is some possibility of under estimation.

There are a number of other factors which could affect the true extrapolation of the miner data to the general public. Most of these are physical considerations, such as the degree of attachment of radioactive progeny atoms to aerosols, the air breathing rate, the degree of radioactive equilibrium and the effect of inflammatory reactionsand the effect of age on lung dosimetry, and will not be dealt with here. The assumption is made either that these parameters will remain fixed or that any changes will be taken into account in models used for extrapolation. An example is the difference in aerosol size distribution and attached fraction noted between mines and normal urban or rural air. This effect can be (and has been, see NCRP 1984a) accounted for by use of a suitable lung model in calculating received doses. In addition, differences in age between the two populations could result in significant differences in lung deposition and clearance. This problem is discussed in a later section. One topic of importance remains, however, that is in the realm of radiobiology. This is the question of the length of the latency period for the expression of genetic damage. If the latency period changes dramatically as a function of dose or dose rate, it will change the final incidence of cancers (due to concurrent competing risks).

Considering the wide variability of this length of time as one moves between species or applies a number of co-carcinogens, it is likely that the problem will be resolved through studies of host factors, and not from purely biophysical considerations. The possibility remains, however, that there are simply different types of biophysical damage, which manifest themselves after different lengths of time. Archer et al. (1971) have studied the epidemiological evidence among miners and have concluded that, for this group, the induction-latency period increases with decreasing dose. They found that this same interval decreased with increasing age of the miner at the start of exposure and with increased smoking habits. They also assessed the effect of dose rate and cumulative dose on the induction-latency period. Increasing the dose rate decreased the period, but cumulative dose (to a given age group had little effect. Similar conclusions were reached by the NCRP (1984a). These data show wide confidence intervals, however, and further studies are in order.

In a comprehensive review of radiation-induced lung cancer, Kennedy and Little (1978), have approached the question of the effect of dose and dose rate on the latency period. They concluded that, in general, this period increases with decreasing dose. For example, Kennedy and Little (unpublished data) found a minimum period of 154 days for the appearance of the first malignant tumor in animals given seven instillations of 0.2 microcuries of ^{210}Po in saline (total dose of 1,850 rad). Compare this to the minimum period of 105 days obtained with 5,000 rad (Little and O'Toole 1974). Of course, one should keep in mind that each form of malignancy (and each species) appears to possess a necessary "minimum" latency period.

The effects of co-carcinogens and promotional agents on the incidence of radiation-induced lung cancer are only poorly known at present. Indeed, due to the complexity of the atmospheres to which both the mining and the general population are exposed, it may be meaningless to refer to the incidence of a purely radiation-induced cancer. Arthur Frank (1978) has recently reviewed causal agents in occupational lung cancer and lists arsenic, asbestos, chlormethyl, ethers, chromium, coal-carbon, iron, steel, nickel, mustard gas and radiation. Di Paolo (1976a) and Di Paola et al. (1976b, 1971) have studied the interaction of radiation with U.V. light, benzo( )pyrene (BP) and a variety of other chemicals and concluded that these co-carcinogens can increase the apparent incidence of radiation-induced carcinomas in hamster cells. Kennedy and Little (1978) showed that protease inhibitors, applied post-irradiation, decreased the incidence of tumors induced by x-rays. It is possible that this effect results from the inhibition of error-prone repair, although this is purely speculative. Terzaghi and Little (1974) have applied BP both before and after irradiation and have noted enhanced incidences in an *in vitro* system, with the enhancement varying according to the schedule of BP administration. The literature abounds with cases in which cocarcinogens and tumor promoters have been shown to affect the incidence of radiation-induced carcinomas, and it would be futile to attempt to cite each study. A general conclusion may be drawn, however; it is highly probable that both the mining and the general population are being subjected to a wide range of co-carcinogens and that this may affect the absolute values of the dose-response curve. As a result, any studies may do best to focus on the relative risk from radiation, where one is interested in the increased risk relative to some "natural," or baseline risk. The true situation probably resides somewhere between relative and absolute risk. Even this approach, however, assumes a small contribution from synergistic agents. If this contribution were large in the miner data, and if the synergistic agents were absent in the environment of the general public, then extrapolation of miner data would overestimate the risk to the populace. Conversely, the occurrence of synergistic agents only in the environment of the general populace would cause an under-estimate. No conclusive data are available to support either contention.

Cigarette smoke has also been suggested as a modifying agent in the induction of lung cancers by radiation. This could be significant due to the fact that smoking was more prevalent in the mining groups than in the general U. S. population (NCRP 1984a). Axelson and Edling (1980), Axelson and Sundell (1978) and Archer (1985) reported that radiogenic lung cancer incidence is somewhat higher in non-smoking miners than in smoking miners, whereas Radford (1981) determined the two rates to be approximately the same. These results are in contrast to earlier studies by Lundin et al. (Lundin 1969), where smoking uranium miners incurred risks of lung cancer that were an order of magnitude higher than the non-smoking miners. This work by Lundin, along with early work by Saccomanno (1971), formed the basis of the Surgeon General's recommendation that cigarette smoke be viewed as a potent co-carcinogen in the induction of lung cancer during exposure to radon progeny. It should be noted that these earlier results (Lundin 1969, Saccomanno 1971) have been modified by the more recent studies due to the convergence of risk in the smoking and non-smoking miner groups. The studies on beagle dogs by Cross et al. (1978) demonstrated that dogs subjected to both cigarette smoke and radon progeny had fewer incremental lung cancers than dogs subjected only to the radon progeny. Cross et al. (1985) have reviewed the data on smoking and radon progeny exposures and conclude that smoking may act to accelerate the appearance of radiation-induced lung carcinomas, although they cite Saccomanno (1982) as challenging this concept. At present, there are insufficient data to assess the impact of smoking habits on the induction of lung cancer by inhalation of radon progeny, a conclusion also reached by the NCRP (1984a), although they suggest that lifetime risk factors for radon progeny exposures in non-smokers may be lower than those developed from the uranium miner data by a factor of 4.

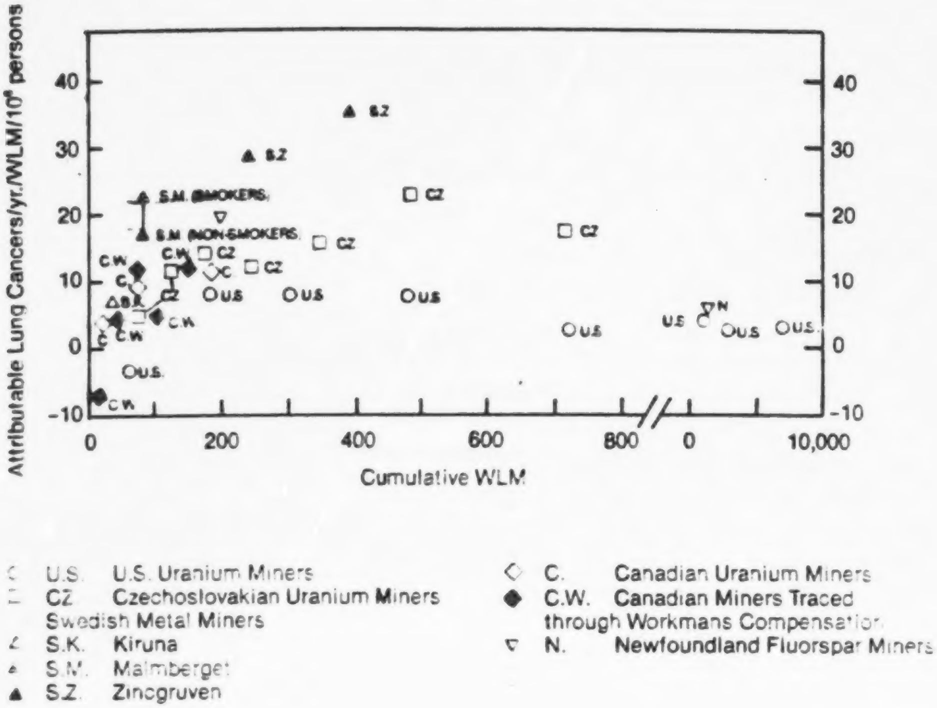
One final consideration remains in the extrapolation of miner data to general environmental exposures. Some studies (Archer 1978) suggested that the effectiveness of radon progeny (per WLM) at inducing lung cancer increases at low doses over that found at high doses. If this is the case, linear extrapolation of the lung cancer rate at high doses to lower doses would not prove to be conservative; instead it would yield an under-estimation of the effects of environmental exposures. Turner and Holoway (1977) have suggested that the lung tumor rate per WLM at low values of WLM in occupational exposures increases by a factor of three or four over that at higher exposure levels, while Archer (1977) reported that this increased effectiveness at low exposures may be as large as a factor of five to ten. These results indicate that, while linear extrapolation of lung tumor rates per WLM from high to low exposures probably will not lead to an over-estimate of the risks from environmental exposures, there is some possibility that the approach will lead to an under-estimate. This factor is not included in existing risk calculations of exposure to environmental levels of radon progeny.

It is now possible to draw a general conclusion concerning extrapolation of ^{222}Rn data from high doses to those typical of environmental conditions. Available evidence suggests that the shape of the dose-response curve is linear or (possibly) supralinear, with no threshold to an exposure at least as low as 30 to 60 WLM (see Figure VII-1). These results suggest a proposition central to this report:

> The induction of lung cancers by relatively low doses of alpha irradiation is supported by experimental, epidemiological and theoretical results. Extrapolation from high-dose data is possible through the use of a linear function, with corrections (perhaps) for variable latency-induction periods.

C. Effect of Age on Risk Factors

All of the earlier discussed risk factors for cancer induction apply strictly to adults, averaged over all adult



Adapted from NCRP 1984a.

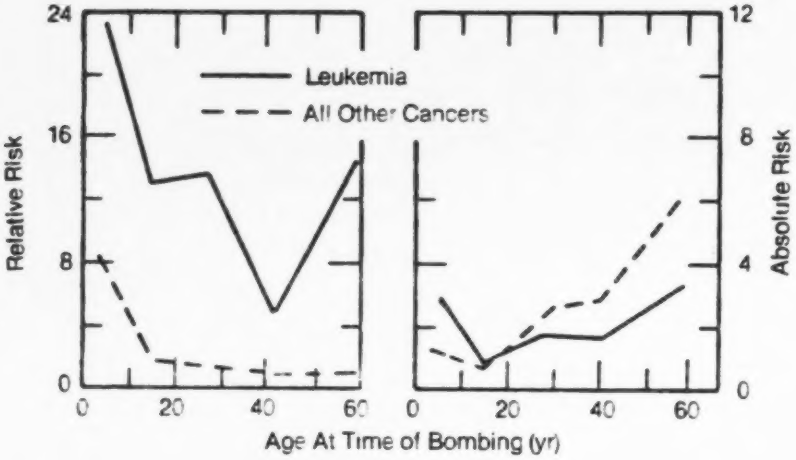
Figure VII-1. Attributable Annual Lung Cancer Risk as a Function of Cumulative Exposure

ages. There is considerable evidence, however, that risk factors are a direct function of age at exposure (Leggett and Crawford-Brown 1983), particularly in the early years of life from neonate through age 18. In a general review of the effects of age on radiosensitivity, Leggett and Crawford-Brown (1983) concluded that the available evidence suggests that age affects the lifetime risk factor for cancer induction, the latency period for radiogenic cancers and the distribution of these cancers in time after exposure. The latency period appears to be dependent upon the type of cancer, with younger ages showing decreased latency periods for both acute and chronic leukemia (relative to adults) and longer latency periods for cancers of other organs. The latter trend is also noted in the uranium miner data (Harley and Pasternack 1981).

The effect of age on lifetime or annual risk factors has not been well elucidated. Results of studies often are conflicting and depend upon the end point chosen and the site of cancer induction. In addition, the trend with age in a specific risk factor depends upon the general form of the risk factor itself. For example (see Figure VII-2), the atomic bomb survivor data suggest that the absolute risk of cancer induction for all cancers except leukemia increases roughly monotonically with age. In contrast, the relative risk coefficient rises dramatically at the younger ages, leveling off at age 20 and then increasing again at age 55 (where the natural rate also begins to rise). The increased risk in age groups older than 40 years at the time of exposure (relative to those in the 20 to 40 age group) has been noted in the uranium miner data (Harley and Pasternack 1981) and is better established than the trend at younger ages.

Unfortunately, while there can be large differences in lifetime risk estimates if calculations of age-dependence are based on the relative and absolute risk coefficients, the present data are not sufficient to permit a choice between the two approaches (see Figure VII-2). In all likelihood, the correct approach incorporates both modeling considerations. If the atomic bomb survivor data are employed to determine relative risk coefficients at all ages (Bee et al. 1978), and if these coefficients continue to apply as the cohort ages, then lifetime risks for lung cancer in the younger age groups will be higher than those in adults by a factor of five to ten (assuming equal doses delivered to the lung mass). On the other hand, absolute risk coefficients in the youngest age groups are lower than those in adult groups for the atomic bomb survivors, so that lifetime risk estimates are higher in the older (>40 year) age groups, assuming equal doses.

Choice between the absolute and relative risk approaches to risk estimation must await further aging of the 1- to 10-year age group of the atomic bomb survivors. Since this age group has only now reached the age of increased "natural" lung cancer rates, the relative risk model predicts that this group will experience a significant increase in absolute risk



Adapted from Leggett and Crawford-Brown 1983.

Figure VII-2 Conclusions Concerning the Differences with Age in Risk from Radiation May Depend Upon Whether the Relative Risk or Absolute Risk Approach is Employed

coefficients in the next 30 years. Until this question is resolved, it appears prudent to perform calculations using both the relative and the absolute risk approaches. Under the relative risk approach, the previously developed risk factor of a 1.4% increase in lung cancer over the natural rate per WLM exposure for adults would increase to as high as 7% to 14% in the 0- to 5-year age group and to approximately 2% to 5% in the 5- to 15-year age group. If these risk factors following exposure at young ages then continue to hold for all years following exposure, the relative risk model predicts lifetime risk coefficients (probability of mortality from cancer induction following exposure at a given age) that are higher than the adult value by approximately a factor of five in the 0- to 15-age group, again assuming equal lung doses within each age group.

D. Lifetime Risks From Radon Exposures

This section is intended to summarize published calculations of the lifetime risk imposed by exposure to radon and progeny. It is assumed that the risk from direct ingestion of radon in water is lower than that from inhalation. As mentioned earlier, this assumption is supported by the calculations of Cross et al. (1983), who found that the lifetime risk from use of water containing 1 pCi/L of radon is highest in the case of lung cancer, lower by at least an order of magnitude for stomach and G.I. tract cancer and lower by a factor of 30 to 75 for whole body doses delivered following ingestion. In addition to reporting previously published results, this section includes the results of calculations performed at Oak Ridge National Laboratories using the relative risk coefficients at various ages.

The ICRP (1981) reviewed available uranium miner data and concluded that a best estimate of the lifetime lung cancer risk following exposure to radon progeny in mines (primarily Czechoslovakian) lies in the region of 1.5 to 4.5×10^{-4}/WLM, depending upon age at exposure. In a similar review that included the Czechoslovakian, U.S. and Canadian exposures, UNSCEAR (1977) determined a lifetime risk of lung cancer in the range of 2 to 4×10^{-4}/WLM. Another review committee sponsored by the NAS (the Committee on the Biological Effects of Ionizing Radiation) used the above data plus the higher results in the Swedish metal miners and Newfoundland fluorspar miners to estimate a lifetime lung cancer risk factor of between 2 and 14×10^{-4}/WLM. The higher value is assumed to hold only in age groups older than 40 at the onset of exposure. Finally, the NCRP in an extensive review of available data, adopted a risk coefficient of 10×10^{-6} per person per year per WLM (NCRP 1984a). If this risk rate is integrated form ages 40 to 85, the lifetime risk is 4.5×10^{-4}/WLM.

Jacobi (1977) determined a value of 2×10^{-4}/WLM as an average lifetime risk of lung cancer for all ages in the mining population and for all mining groups. This agrees well with the most recent modeling efforts by Harley and Pasternack (1981) and RPC (1980), who have estimated a lifetime risk coefficient of between 1 and 2 x 10^{-4}/WLM. Since this last modeling effort is the most complete to date and forms the basis for the recommendations of the National Council on Radiation Protection and Measurements in their report on evaluation of occupational and environmental exposures to radon and radon progeny, the Harley-Pasternack model forms the prelimary focus of discussion here. An important point to bear in mind is that only the Harley-Pasternack (HP) model incorporates considerations of the risk incurred by radon progeny exposures in infants and children.

The HP model incorporates several assumptions suggested by a review of the uranium miner data. The authors assume that lung cancer does not occur during a latent period that extends a minimum of five years following exposure. In addition, the latent period is such that no cancers will appear until the exposed individual reaches 40 years of age. Another model assumption is that there is repair of damage to the bronchial epithelium, characterized by a repair half-time of 20 years. This repair factor apparently was introduced to account for the temporal pattern of lung tumors in the uranium miner population and for the increased risk in the 40+ age group relative to the less than 40 year-age group. If this assumption is incorrect, the result would be a significant under-estimate of the lifetime risk from exposure at very young ages. Finally, the model assumes that competing risks should be included through the use of a life table (World Health Organization 1978) and employs an environmental exposure-to-dose conversion factor of 0.7 rad/WLM for all age groups. As noted earlier, this conversion factor may prove to be too low at very young ages (0 to 15 years at time of exposure), with possible increases by a factor of two to three in this younger age group if the results of Hofmann (1982a) were to be used. Consideration of the increased risk at young ages, however, does not greatly change the lifetime risk for a newly born cohort (Harley and Pasternack 1981), since this lifetime risk from continous exposure changed by only 10% when age-dependent or age-invariant exposure-to-dose conversion factors were employed.

In their recent report Cross et al. (1983) have summarized the results of calculations for environmental exposures performed using the above model. They assumed an equilibrium factor of 0.7, an unattached RaA/Rn ratio of 7%, 16 hours/day of activity and 8 hours/day of rest and an age-invariant exposure-to-dose conversion factor of 0.7 rad/WLM. For continuous exposures beginning at 1 year of age, they determined a lifetime lung cancer risk of 9.1 x 10^{-3}/WL M/year under environmental conditions. Expressed in units of air concentration, this lifetime lung cancer risk corresponds to 3.6 x 10^{-3}/pCi/L in the dwelling air (the concentration refers to the radon). With a water-to-air conversion factor of 10^{-4}, this latter risk would yield $3.6 \times 10^{-3}/10^{4}$ pCi/L of ^{222}Rn in water used in the home, or 3.6×10^{-7}/pCi/L of ^{222}Rn in the water. These values may be compared with the lifetime risk of lung cancer among non-smokers in the U.S., which has been estimated by Garfinkel (1980) and Enstrom and Gadley (1980) to reside in the neighborhood of 0.6%. Under the Harley-Pasternack model, 1 pCi/L of radon in household water would, then, increase the lifetime risk of lung cancer by 0.006% over that expected in the absence of radon in water.

The Cross report (1985) also presents the results of calculations on populations with the age distribution currently characterizing the U.S. (World Health Organization 1978). In this instance, the lifetime lung cancer risks for lifetime exposures will be lower than those imposed on a newborn cohort, since exposures begin at various ages which are distributed in frequency according to the frequency of that age in the U.S. population. These calculations yield lifetime risks from environmental exposures of 5.6×10^{-3}/WLM/year, or 2.1×10^{-3} per pCi/L of ^{222}Rn in air. Again, using a water-to-air conversion factor of 10^{-4}, the latter risk coefficient corresponds to a lifetime risk of 2.1×10^{-7}/pCi/L of ^{222}Rn in the water used in a dwelling.

In addition to the calculations performed by Cross et al. (1983), several lifetime risk calculations were performed at Oak Ridge National Laboratory for the purpose of this report.

These calculations also utilized a life table methodology (Sullivan et al. 1981), but employed relative risk coefficients of 1.4% per WLM as reported by Walsh (1979) for adults. In addition, the calculations were performed using age-dependent exposure-to-dose conversion factors published by Hofmann (1982a). Since the Hofmann report contains calculations of this factor using an assumption of both age-invariant basal cell depth and age-dependent basal cell depth, the present calculations have been performed using both sets of the Hofmann data. Averages of the Hofmann data were obtained from figures in his paper (1982a or see Figure III-6); the cumulative exposure-to-dose conversion factor in the 0 to 5 age group was determined to be higher than that in the adult age group by a factor of 1.25 when basal cell depth is age-invariant and by a factor of 2.1 when basal cell depth varies with age. Similarly, the exposure-to-dose conversion factor for the 5 to 15 age group was increased over the adult value (0.7 rad/WLM) by a factor of 1.5 when basal cell depth is age-invariant and by a factor of 2.0 when basal cell depth varies with age. In both cases (age-invariant and agedependent basal cell depth), the risk factor in the 0 to 15 age group was increased by a factor of five over that in the adult group (which was 1.4% per WLM). A summary of the calculational results is contained in Table VII-1. These results may be compared directly with those obtained using the Harley-Pasternack model as presented in Cross (1985) and as reported earlier in this document (3.6×10^{-7} per pCi/L). Clearly, use of the age dependent relative risk coefficient increases lifetime risk slightly over that obtained using the absolute risk approach of Harley and Pasternack (1981).

Table VII-1 Lifetime Risks of Lung Cancer Using a Relative Risk Model

Exposure is continuous to 1 pCi of Rn-222 per Liter of water

Latency Period (years)	Plateau (years)	Child (0-15 year) Risk (%/rem)	Adult (>15 year) Risk (%/rem)	Lifetime Risk
5	30	0.2	0.2	3.27×10^{-7}
5	110	0.2	0.2	7.28×10^{-7}
5	30	1.0	0.2	3.56×10^{-7}
5	110	1.0	0.2	1.64×10^{-6}

Assumes $Ca/Cw = 10^{-4}$

VIII. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

The quantification of toxicological effects of a chemical consists of an assessment of the non-carcinogenic and carcinogenic effects. In the quantification of non-carcinogenic effects, an Acceptable Daily Intake (ADI) is calculated. From this, an Adjusted Acceptable Daily Intake (AADI) and Health Advisory (HA) values for the chemical are calculated to define the appropriate drinking water concentrations to limit human exposure. For ingestion data, this approach is illustrated as follows:

$$\text{ADI} = \frac{(\text{NOAEL or LOAEL in mg/kg/day})(\text{Body Weight in kg})}{\text{Uncertainty/Safety Factor}}$$

$$= \text{mg/day}$$

$$\text{AADI} = \frac{\text{ADI}}{\text{Drinking Water Volume in L/day}} = \text{mg/L}$$

where:

NOAEL = no-observed-adverse-effect level.

LOAEL = lowest-observed-adverse-effect level.

Body weight = 70 kg for adult or 10 kg for child.

Drinking water volume = 2 L per day for adults or 1 L per day for children.

Uncertainty/Safety Factor = 10, 100 or 1,000.

Utilizing these equations, the following drinking water concentrations are developed for non-carcinogenic effects:

1. A one-day HA for 10-kg child.
2. A one-day HA for 70-kg adult.
3. A ten-day HA for 10-kg child.
4. A ten-day HA for 70-kg adult.
5. A lifetime AADI for a 70-kg adult.

The distinctions made between the HA calculations (items 1 through 4) are associated with the duration of anticipated exposure. Items 1 and 2 assume a single acute exposure to the chemical. Items 3 and 4 assume a limited period of exposure (possibly 1 to 2 weeks). The HA values will not be used in establishing a drinking water standard for the chemical. Rather, they will be used as informal scientific guidance to municipalities and other organizations when emergency spills or contamination situations occur. The AADI value (Item 5) is intended to provide the scientific basis for establishing a drinking water standard based upon non-carcinogenic effects.

A NOAEL or LOAEL is determined from animal toxicity data or human effects data. For animal data, this level is divided by an uncertainty factor because there is no universally acceptable quantitative method to extrapolate from animals to humans. The possibility must be considered that humans are more sensitive to the toxic effects of chemicals than are animals. For human data, an uncertainty factor is also used to account for the heterogeneity of the human population in which persons exhibit differing sensitivity to toxic chemicals.

An Office of Drinking Water (ODW) modification of the guidelines set forth by the National Academy of Sciences (NAS 1977, 1980) is typically used in establishing uncertainty factors as follows:

- An uncertainty factor of 10 is used when good acute or chronic human exposure data identifying a NOEL/NOAEL are available and are supported by acute or chronic toxicity data in other species.
- An uncertainty factor of 100 is used either when good acute or chronic toxicity data identifying a NOEL/NOAEL are available for one or more animal species (but human data are not available), or when good acute or chronic toxicity data identifying a LOAEL in humans are available.
- An uncertainty factor of 1,000 is used either when limited or incomplete acute or chronic toxicity data are available, or when good acute or chronic toxicity data identify a LOAEL (but not a NOEL/NOAEL) for one or more animal species.

The uncertainty factor used for a specific risk assessment is judgmental. Factors that cannot be incorporated in the NAS/ODW guidelines for selection of an uncertainty factor, but must be considered include: (1) the quality of the toxicology data; (2) the significance of the adverse effect and; (3) the existence of counterbalancing beneficial effects.

If toxicological evidence requires the chemical to be classified as a potential carcinogen (there is evidence to classify radon as a carcinogen following ingestion), mathematical models are used to calculate the estimated excess cancer risks associated with the ingestion of the chemical via drinking water. The bioassay data used in these estimates are from animal experiments. In order to predict the risk for humans from these data, it must be converted to an equivalent human dose. This conversion includes correction for non-continuous animal feeding, non-lifetime studies and for the difference in size. The factor that compensates for the size difference is the cube root of the ratio of the animal and human body weights. It is assumed that the average human body weight is 70 kg and that the average human consumes 2 liters of water per day. The multistage model is then fit to the equivalent human data to estimate the risk at low doses. The upper 95% confidence limit of this estimate is used. Excess cancer risks can also be estimated using other models such as the one-hit model, the Weibull model, the logit model and the probit model. There is no basis in the current understanding of the biological mechanisms involved in cancer to choose among these models. The estimates of low doses for these models can differ by several orders of magnitude.

The scientific data base used to calculate and support the setting of risk rate levels has an inherent uncertainty. This is because the tools of scientific measurement, by their very nature, involve both systematic and random error. In most cases, only studies using experimental animals have been performed. There is thus uncertainty when the data are extrapolated to humans. When developing risk rate levels, several other areas of uncertainty exist, such as (1) incomplete knowledge concerning the health effects of contaminants in drinking water; (2) the impact of test animal age, sex and species and the nature of target organ systems examined on the toxicity study results and; (3) the actual rate of exposure of internal targets in test animals or humans. Dose-response data are usually only available for high levels of exposure, not for the lower levels of exposure for which a standard is being set. When there is exposure to more than one contaminant, additional uncertainty results from a lack of information about possible synergistic or antagonistic effects.

A. Non-Carcinogenic Effects

The knowledge base for non-carcinogenic effects is for low-LET radiation. There is an absence of information on non-carcinogenic effects relating to high-LET radiation such as the alpha particle radiation from naturally-occurring isotopes such as radon (^{222}Rn). In any case, it would appear that the most important effects are the carcinogenic ones.

Besides cancer, the only other known health effects likely to occur due to internally deposited radionuclides include non-stochastic effects, genetic effects and in utero developmental effects.

In general about 10 rad or more of low Let radiation is needed to induce non-stochastic effects. This threshold is well above the levels of environmental exposure due to radon ingested from drinking water.

Using a conservative estimation approach, the doubling dose (dose needed to double the risk) for genetic effects is 100 rad (USEPA 1984) if the dose is delivered to the ovary or testis. Since little radon is deposited in the reproductive organ, the genetic effects would be expected to be negligible compared to bone cancer.

The rate of teratogenic effects is in the range of 7.4×10^{-3} to 5.0×10^{-4} cases of mental retardation per live birth per rad of low LET radiation delivered between weeks 8 and 15 of gestation, with no threshold identified at this time (USEPA 1984). This effect is negligible in comparison to that due to bone cancer.

B. Quantification of Non-Carcinogenic Effects

In the absence or appropriate dose-response data for non-carcinogenic effects, it is not possible to derive an AADI for radon (^{222}Rn).

C. Carcinogenic Effects

The potential human health consequence of inhaling radon is lung cancer. This has been demonstrated by experience with uranium miners. The evidence clearly shows a dose-response relationship between radon levels in uranium mines and lung carcinomas.

Human epidemiologic data obtained from groups of underground matal-ore miners in Czechoslovakia, Canada, the United States, Sweden, Britain and Newfoundland are currently available. These data have been summarized and reviewed by many organizations and the authors (NIOSH 1971, NAS 1972, NAS 1980, Jorgensen 1972, Snihs 1973, Renard 1974, deVilliers and Windish 1964, Wright and Couves 1977, Evans et al 1981). Table VIII-1 displays a summary of results as published by Walsh (1979). Some of this data is shown graphically in Figure VIII-1.

From the published data, absolute risk values of between 1.5 and 50 cancers/WLM/year/10^6 persons may be noted. The NCRP (1984) has reviewed available epidemiologic data and calculated a mean risk factor of 12 cancers/year/WLM/10^6 persons. They also reported a standard deviation of two for this estimate, although this should be viewed as extremely optimistic, since it ignores problems of measurement error which exist in all of the studies reviewed.

Table VIII-1 Excess Relative Risk (in Percent) of Radiogenic Lung Cancer in Groups Exposed to Alpha Particles, x-rays, gamma-rays and Neutrons

Exposed group	Type of radiation	Percent Increase in Excess Relative Risk		
		per WLM	per rad	per rem
Uranium miners				
U.S. (white)	alpha	0.9		
U.S. (Indian) WLM > 300	alpha	0.4		
U.S. (15 years after start), WLM < 500	alpha	0.9		
Canada	alpha	0.9		
Czechoslovakia	alpha	1.2		
Fluorspar miners	alpha	3.0		
Metal miners	alpha	0.5		
Swedish metal miners	alpha	3.2		
Thorotrast (Portugal)	alpha		1.0	
Bomb survivors	gamma		0.3	0.3
			1.0	0.2
Spondylitics	x-rays		0.2	0.2

D. Quantification of Carcinogenic Effects

The effective dose equivalent rate and risk rates due to inhaled radon are derived from values from ICRP Report #32 (1981).

The lifetime lung cancer mortality associated with radon in drinking water is calculated as

$$r = a_1 \times a_2 \times y_1 \times y_2 \times y_3 \times y_4$$

where

a_1 = the number of years of exposure = 70 years (the average lifetime).

a_2 = the number of WLs corresponding to a concentration of 100 pCi/L_a of ^{222}Rn in secular equilibrium with its short-lived progeny = 1 WL/100 pCi/L_a, where L_a is liters of air.

y_1 = the ^{222}Rn equilibrium factor = 0.3 to 0.7 (to compensate for the lack of equilibrium).

y_2 = equivalent occupational working months per year for a member of the population = (12 to 24) months/year; this allows for variation in residency time and breathing rate.

y_3 = transfer factor for radon from water to air = (0.17 to 3.5) x 10^{-4} (USEPA 1984).

y_4 = lifetime lung cancer mortality risk for exposure of 1 WLM = (1.5 to 4.5) x 10^{-4}/WLM (ICRP 1981).

Assuming that the errors propagate geometrically, the combined value is in the range of 2.00×10^{-8} to 59.7×10^{-8} excess cases of lung cancer/pCi/L_w (where Lw is liters of water).

The calculations of effective dose equivalent involve forming the products of quantities whose uncertainties were expressed in terms of a range. In general, these ranges are not determined by knowing the functional distribution, but are estimates (often rough estimates) of the uncertainty. In order to propagate these uncertainties when the quantities are combined, however, the form of the distribution needs to be either determined or assumed.

One distribution that has been widely applied to environmental parameters is the log normal distribution. In this distribution, the logarithms of variables are normally distributed.

The range in the dose equivalent factor for radon is from 0.031 to 0.033 Sv/WLM (ICRP 1981), and the weighting factor is 0.21, derived from BEIR III (NAS 1980). Thus, the effective dose equivalent is of the form

$$d = a_1 \times a_2 \times a_3 \times y_1 \times y_2 \times y_3 \times y_4$$

where

a_1 = number of WLs levels corresponding to a concentration of 100 pCi/L_a of ^{222}Rn in secular equilibrium with its short-lived progeny = 1 WL/100 pCi/L_a.

a_2 = 100 rem/Sv.

a_3 = 1,000 mrem/rem.

y_1 = the ^{222}Rn equilibrium factor; range 0.3 to 0.7.

y_2 = equivalent occupational working months per year for a member of the population = (12 to 24) months/year; this allows for variation in the residency time and breathing rate.

y_3 = transfer factor from water to air; range (0.17 to 3.5) x 10^{-4} (USEPA 1984). The factor is in units of pCi/L of air/pCi/L of water.

y_4 = mean effective dose equivalent factor from ICRP 32 using the BEIR III weighting factor. The ICRP 32 lung model mean dose equivalent for the pulmonary region is from 0.031 to 0.033 Sv/WLM, and the BEIR III weighting factor is 0.21. This gives a range of 0.0065 to 0.0069 Sv/WLM. In order to allow for the uncertainties, it is estimated that the range of this factor is from 0.004 to 0.01 Sv/WLM.

Then, combining the error ranges by summing them as geometric standard deviations (USEPA 1980) yields

$$z = (0.8 \text{ to } 20) \times 10^{-3} \text{ mrem/year/pCi/L}_w$$

This risk and the annual effective dose equivalent (EDE) for inhaled ^{222}Rn have been estimated using the data from ICRP Report 32. The estimates, as described in Section X.B, are tabulated in Table VIII-2 for different risk levels. The numerical estimates in Table VIII-2 are rounded off to fit on such a table for all radionuclides. The discrepancy of a factor of two between Section VIII.B and Table VIII-2 is within the overall uncertainty of a factor of four to five.

Table VIII-2 Lifetime Risk and Annual Effective Dose Equivalent Rates as They Relate to Concentrations of Radon in Drinking Water[a]

Lifetime individual risk	Annual effective dose equivalent rate (mrem/year)	Drinking water concentration[b] (pCi/L)
10^{-3}	100	10,000
10^{-4}	10	1,000
10^{-5}	1	100
10^{-6}	0.1	10

[a] Assuming an ingestion rate of 2 L/day.
[b] Rounded off to one significant figure and involving an uncertainty of a factor of four to five.

E. Existing Standards

Table VIII-1 lists standards from other countries in the world.

Table VIII-3 Standards or Guidelines for Radon in Drinking Water in the World

Country	Standard or guideline for ^{222}Rn
Kuwait	30 pCi/L (1.1 Bq/L)
USA	2,000 pCi/L (National Bureau of Standards, Handbook 69)

Adapted From Survey of the Uranium Task Force, Dept. of Health, Province of Nova Scotia, Canada (1984).

F. Special Considerations

Radon is found in groundwater supplies, and although the levels are high in certain geographical regions (e.g., the northeast U.S.), it is generally found in water supplies throughout the country.

There is no clear evidence of populations that are specifically sensitive to radon toxicity due to inhaled radon, but there is a suggestion of synergism with cigarette smoke.

G. Quantification of Cancer Risks

This risk and the annual effective dose equivalent (EDE) for inhaled ^{222}Rn have been estimated using the data from ICRP Report 32. The estimates, as desribed in Section X.B, are tabulated in Table VIII-2 for different risk levels. The numerical estimates in Table VIII-2 are rounded off to fit on such a table for all radionuclides. The discepancy of a factor of two between Section VIII.B and Table VIII-2 is within the overall uncertainty of a factor of four to five.

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APPENDIX A

UNCERTAINTIES IN RISK ESTIMATES FOR USE OF RADON-LADEN WATER IN THE UNITED STATES

Calculations of the health effects that accrue from the use of water obtained from a source containing radon are beset by areas of uncertainty, with the nature and level of the uncertainty being dependent upon the particular question being asked. As is true in most instances of uncertainty analysis, the level of confidence placed on specific statements decreases as the statements pertain to progressively more specific populations. In other words, statements about the average risk applicable to a population will be characterized by lower levels of uncertainty than are statements about the risk to a solitary person in that population. In fact, under the long-term frequentist approach to statistics (von Mises 1957), probabilistic statements are not even applicable to individuals, but only to populations of those individuals.

The following development of uncertainties presumes that the concept of uncertainty in a statement corresponds to the degree to which that statement is supported by past experience and knowledge, relative to any competing statements. As such, the definition of uncertainty used here corresponds to the Bayesian conception for statistics (Win 72). It is assumed that the intent of uncertainty analysis is to determine all possible inferences consistent with existing data and conceptual schemes, followed by some assignment of the degree of belief associated with each specific inference. The result is a relative ranking of possible inferences (here, of health risk), with both qualitative and quantitative information playing a role in assignment of uncertainties. No metaphysical uncertainties are examined in the present report, since the applicability of past experience to inferences of future experience is taken for granted in scientifically derived predictions of the effects of an action.

In general, uncertainties can arise at any stage in the process of drawing an inference. A statement is inferred from a set of past experiences. It is often the case that competing sets of past experience are available for the drawing of inferences, with no single set possessing complete logical dominance over the competitors. As a result, it is never certain which set of experiences ought to be used in drawing inferences. Even if there were only one available set of experiences, theoretical explanations of the relationship between those experiences and the problem-at-hand are usually available in many forms. Theories must, in turn, be translated into mathematical models relating the theoretical terms to empirically observable properties of the system being studied. There are usually several competing mathematical models, each more or less consistent with existing data and theories.

Finally, the values to be assigned to parameters in any given model will be uncertain due either to small sample sizes in past experience or to some form of measurement error. Each of the above forms of uncertainty is discussed below as it appears within the context of obtaining risk estimates for radon-laden water use in dwellings.

No calculation of risk can proceed until the concentration of radon in water has been determined. If the calculated risk is to be specific to an individual, the radon concentration in water used by the individual must be determined. As noted in the section on occurrence of radon in water (see Table IV-6), water supplies in a state display a large range of values. Measured radon concentrations within a state and within a population size (see Table IV-5) are characterized by log normal distributions (Aitchison and Brown 1957), with a geometric standard deviation of approximately three. This suggests that use of the state-average radon water concentration in calculating risks to an individual could result in estimates that deviate from the correct value by a factor of three at the 68% confidence level (a factor of nine at the 95% confidence level).

This assumes, of course, that the measured distribution of water samples drawn from a state are representative samples of all water supplies in the state. While the measurement values reported in Tables II-2, IV-4, IV-5 and IV-6 of this report may be representative for the larger water supplies (serving populations of greater than 1,000), the radon content of water in smaller sources is only poorly determined at present, with most states having less than ten samples analyzed in the less-than-1,000 population groupings. The radon concentration in private wells is only very poorly known, with essentially no data in more than half of the states. Since these wells are probably characterized by a much higher radon concentration than is found in other sources, the uncertainty in radon concentration in these wells can have significant impact on regulatory concerns. Given the fact that the available samples in the less-than-100 group of Table IV-7 contain data obtained from water sources suspected of high radon levels prior to sampling, and given the large variance associated with these samples (G.S.D. >3), it appears likely that the mean radon concentration in private wells in the U.S. may be currently under- or over-estimated by a factor of three or more, with a reasonable upper bound being in the neighborhood of a factor of ten. (Since the samples may be biased toward high radon concentrations, it is probably more likely that the mean is over-estimated, rather than under-estimated.) Use of the current U.S. average radon concentration in private wells (<100 people served) to determine risk to an individual could be expected, therefore, to result in errors easily as large as an order of magnitude.

Errors in the actual measurement of radon in water (such as the measurement data used for Figure IV-4 of the present report) should not greatly influence the current calculation of the distribution of radon concentration in water sources in the U.S. This is because measurement errors are very small compared to the variability of mean radon concentration between different sampling sites or sources of water. There is some possibility, however, that reported measurements of radon concentration in water may be too low due to loss of radon during sampling and measurement. This potential error should not be large, but it will be systematic for all samples (since radon can be lost from a sample, but not artifically increased). Given the state of control over water sampling methods, it is unlikely that current measurements are in error by more than 25%. This estimate includes the measurement error introduced by counting statistics and fluctuations in the electronics and calibration associated with measurement procedures.

In summary, measurements of the radon concentration in water at a source should be accurate to within 25% (95% confidence level), assuming several measurements are made. Use of the state average (for a specific population size) in estimating radon water concentration in an unsampled source should be accurate to within a factor of nine (95% confidence level). Use of current data to determine the mean radon concentration in U.S. water supplies should be accurate to within a factor of three or four (95% confidence level), with estimates of the mean radon concentration in private wells being accurate only to within a factor of ten of the correct value (95% confidence level). It must be borne in mind that these are only reasonable estimates of the associated uncertainties, being the result of judgments made in light of available data on measurement errors and source variability.

Given an accurate and precise determination of the concentration of radon in a dwelling, it is necessary to determine the resulting radon progeny concentration in the air of the dwelling. The ability to determine this quantity will depend upon the extent to which the factor, C_a/C_w, can be estimated. As demonstrated in earlier sections of this report, calculated values of C_a/C_w depend critically on assumed values of dwelling ventilation rates, building size, water uses and water temperature. Calculated values of this factor range from 1×10^{-5} to 20×10^{-4} (Gesell and Prichard 1980, UNSCEAR 1977, UNSCEAR 1982, Kahlos and Asikainen 1980), with a mean of approximately 1×10^{-4}. The same mean has been determined from experimental measurements performed directly in homes using radon-laden water. Samples obtained within the U.S. indicate values of C_a/C_w that range from 1×10^{-5} to 10×10^{-4}. Examination of both sets of data (calculation and measured) indicates that the mean value for C_a/C_w in dwellings in the U.S. is approximately 1×10^{-4}, with an uncertainty in the neighborhood of a factor of two to three (95% confidence level). The reader is referred to Tables IV-10 and IV-11 for a review of the data used in this assessment. Values of C_a/C_w for a specific home could deviate from the mean value by as much as an order of magnitude.

Since the radon itself is not of radiological concern, it is necessary to estimate the radon progeny concentration in dwelling air, given a specific radon concentration. As described earlier, this radon progeny concentration is calculated through use of an estimated equilibrium factor. As reported in the Introduction, this equilibrium factor, F, is a function of the ventilation rate and aerosol concentration in a dwelling. Measured values of F in dwellings range between 0.25 and 0.8 (Steinhausler et al. 1980, Swedjemark and Hakansson 1978, Swedjemark et al. 1979, Letourneau et al. 1978, Strain et al. 1979, Makelainen 1980, UNSCEAR 1982), with various authors employing values between 0.5 and 0.7 for purposes of risk estimation (Cross et al. 1985). It appears unlikely that use of a mean value of 0.5 in calculations will result in errors of greater than 50% (95% confidence level). This source of error should be small, compared to other sources outlined below, since Cross et al. (1985) have demonstrated that changing the factor, F, from 0.7 to 0.55 results in less than 10% change in estimated lung dose.

Once the radon progeny concentration has been determined, it is necessary to divide the concentration into the components of attached and unattached progeny. This separation is necessary due to the large differences in tracheobronchial deposition for these two fractions. The calculation of dose delivered to any subpopulation of lung cells is a strong function of this degree of attachment, being primarily responsible for the increased exposure-to-dose conversion factor calculated for environmental exposures relative to mining exposures. As indicated in Figures II-5 and II-6 of the present report, the uncombined fraction changes by a factor of five as the aerosol concentration varies by an order of magnitude. Fortunately, large variations in the unattached fraction are not found (Cross et al. 1985) in the environment. In light of the fact that the difference between unattached fractions found in uranium mines and in normal homes is small (4% and 7%, respectively), and despite large differences in environmental conditions, it appears that use of a mean value of 7% in U.S. dwellings should not be in error by more than 50% (i.e., 95% confidence interval of 4% to 10%). Using the lung deposition model of Crawford-Brown (1982), which is similar to the model employed by other authors for calculating doses from radon progeny exposures, a change in unattached fraction from 4% to 10% for 0.1-micron diameter particles should result in a dose to basal cells of the epithelium that changes by 65%. As a result, use of a mean value of 7% for the unattached fraction should result in dose estimates to critical cells that are accurate to within 50% or less. This uncertainty applies to consideration of the accuracy with which the mean lung dose to the U.S. population can be calculated, assuming that only the unattached fraction is uncertain.

Lung model calculations are also affected by assumed breathing rates, anatomical model, aerosol size distribution, etc. Breathing rates themselves are functions of the activity assumed to characterize the lifestyle of the exposed population. Anatomical models are available in many forms, as outlined in earlier sections of this report. In general, the more anatomically correct models are more difficult to employ in calculations. The interactions between various modeling parameters, and their ultimate effect on calculated lung doses, have not been determined at present. There does exist, however, a set of exposure-to-dose conversion factors calculated by different authors using different assumed values for each model parameter (Harley and Pasternack 1981, Hofmann 1982a,b, Jacobi and Eisfeld 1980, James et al. 1980, 1981, Altshuler et al. 1964, Jacobi 1964). These calculated values of the conversion factor range from 0.3 to 1.0 rad/WLM. Included in this variability is the effect of moving the location of the critical cells from a uniform depth of 22 micrometers below the mucus surface to the distribution reported by Gastineau et al. (1972). Variability would be even lower if the mean dose to the bronchial epithelium were calculated, rather than the dose to the basal cells. In light of these considerations and others given earlier, an assumed exposure-to-dose conversion factor of 0.7 rad/WLM should not be in error by more than a factor of two (95% confidence level), even when other considerations, such as variability in the assumed distribution of radon progeny in the mucus layer, are incorporated.

This uncertainty applies only to the calculation of the mean exposure-to-dose conversion factor for an entire population. Values specific to individuals may differ greatly from the mean due to differences in breathing patterns and anatomical structure. Hofmann (1983, 1982), Cuddihy (1979) and Brown and Taylor (1975) have studied empirically the variability in individual-specific deposition following exposure to a radioactive aerosol. They found that deposition (and dose) varies lognormally about the median value, with a geometric standard deviation of between three and four. As a result, even if the correct median value for the exposure-to-dose conversion factor could be specified for a population, individuals in that population could have doses that deviate from this median by factors of between 9 and 16 (95% confidence level).

Summarizing the above discussion, current estimates of the mean value for the dose delivered to bronchial epithelium from use of a specified amount of water in a home, assuming a known radon-in-air concentration, should not be in error by more than a factor of two to three. Actual values for individuals in the population could be in error by as much as an order of magnitude (95% confidence level). If the air concentration is not known (which is more typical), then specification of the radon water concentration should result in estimates of the mean lung dose that are not in error by more than a factor of between three and five for the U.S. population (95% confidence level), while doses to individuals could be in error by as much as a factor of 20 to 30 (95% confidence level). This large variability is due to individual-specific differences in the ratio, C_a/C_w, and in deposited fractions in the lung.

Each of the above uncertainties relates to variability introduced into model calculations through measured variability in model parameters. Another source of uncertainty may be found in the inability to present theories of lung cancer to specify the critical cells within the lung mass and their location within the various generations of the lung. As mentioned earlier, current estimates of lung doses from environmental exposures to radon progeny employ the assumption that the basal cells of the bronchial epithelium are the sites of carcinomas. While the evidence for this assumption is fairly strong, being in line with current radiobiological thought relating radiosensitivity to cell turnover rates and state of differentiation, the possibility remains that cancers arise from irradiation of the pseudostratified columnar epithelium or from interactions between irradiated epithelia and basal cells. Calculated doses to critical cells can change appreciably as the location of those cells changes with respect to the mucus surface (see Figure III-2 in the present report). In addition, it is not clear at present whether specific generations of the lung are more highly associated with cancer induction by radon progeny, with exposure-to-dose conversion factors differing between generations.

Fortunately, this lack of specificity in the location of critical cells is not as significant as it might appear at first glance. Risk factors (risk per rad or risk per WLM) for environmental exposures to radon progeny are obtained from the uranium miner data. While absolute values of the dose delivered to the lungs of uranium miners can differ appreciably from those expected in environmental exposures, the relative values of doses to various cellular subpopulations within the lung do not change greatly. As a result, the dose per unit exposure for the "correct" critical cellular subpopulation should be a linear function of the dose to any other subpopulation within range of the alpha emissions in the tracheobronchial region. Risk factors derived from the mining exposures, therefore, will apply to environmental exposures (following suitable correction for exposure-to-dose conversion factor differences) as long as doses to the same cellular subpopulation are computed in each case. Unfortunately, potential effects of changing levels of co-carcinogens cannot be determined at present.

The risk factors (risk per rad or risk per WLM) for the mining populations have been calculated for many mining groups. Despite large potential differences in these groups (including differences in potential co-carcinogens or synergistic agents), the risk estimates are fairly stable. Reference to the Health Effects Section of the present report will indicate that calculated absolute risk factors range between 1.5 and 50 cancers/WLM/year/10^6 persons. The mean value of 10 x 10^{-6}/ person/WLM/year, therefore, is within a factor of five of the upper and lower bounds, with most of the risk factors falling within a factor of two to three of this mean value. It seems reasonable, therefore, to assume that the mean value in the literature is correct to within a factor of two to three at high doses or exposures (95% confidence level), assuming all of the excess lung cancers in the mining populations were the result of exposure to radon progeny. Similar uncertainties should apply to relative risk estimates (see Table VI-2).

It is not currently possible to place quantitative estimates on the uncertainties associated with extrapolation of miner risk factors to lower exposures. As indicated by Figure VII-1, either a quadratic, linear or linear-quadratic curve may be fit to available data, although even the best fitting quadratic curve is well approximated by a linear function over the range of measured exposures. The existence of a threshold for lung cancer induction has never been proven or disproven and remains a rather weak possibility at present.

Counteracting the potential for a threshold is the evidence suggesting that the effectiveness of radon progeny at inducing lung cancer (risk per WLM) may actually increase at low exposures (Archer 78), resulting in a supralinear exposure-response curve. If the latter possibility holds true, available evidence (Archer 78) suggests that risk factors obtained from the mining data may be too low by a factor of five to ten. The net result of these considerations is that risk estimates obtained from mining data (10 x 10^{-6}/year/person/WLM) and applied to environmental exposures could be too low by an order of magnitude or too high by factors of three to five (assuming no threshold) or infinity (if a threshold exists above environmental levels of exposure). Unfortunately, no further quantification of the uncertainties underlying extrapolation of the miner risk estimates can be given at present. As a side note, the uncertainties listed above should be interpreted as including uncertainties introduced by incomplete follow-up of the exposed populations used in the epidemiological studies, since this source of uncertainty should be small compared to the other sources.

Calculations of lifetime risk following the onset of exposure to radon progeny are beset by two further uncertainties. The first is the latency period and plateau period associated with lung cancer induction. In one of their reported risk models, Harley and Pasternack (1981) varied the latency interval from 5 to 20 years, with a constant rate of

50×10^{-6} cancers/person/year/WLM applying after the latency interval. The lifetime risk for persons 20 years old at the first exposure was not affected by choice of latency period, while the lifetime risk to persons aged 60 decreased by a factor of seven as the latency was increased from 5 to 20 years. Only small changes were evident at any age (maximum 30%) when latency was changed from five to ten years. As a result, uncertainties in the latency period result in uncertainties in lifetime risk that are on the order of 10% to 30% for a typical age distribution in a population.

There is little evidence of a finite plateau period for lung cancers. The rate, however, may not be constant in time following the end of the latency period. Harley and Pasternack (1981) have calculated the lifetime risk in exposed persons, assuming varying rates of decrease of the height of the peak during the plateau period. Their results indicate that changes in this rate of decrease over a reasonable range ($t_{1/2}$ changes from 10 to 40 years) can result in lifetime risk estimates that change by as much as a factor of four if exposure is at age 20, and by a factor of two if exposure is at age 60.

Considerations of latency period and plateau period are particularly significant when exposures begin at neonatal ages. Assumption of a decreasing plateau height with time after exposure, or latency interval, can result in significant decreases in risk predicted for the youngest ages. Harley and

Pasternack (1981) employed this approach to account for the increased risk in the 40+ age group of uranium miners relative to the less-than-40 age group. The net result of their modeling assumptions is that lifetime risk per WLM of exposure generally decreases as age-at-exposure decreases to childhood ages. Employing this model (absolute risk coefficient decreasing with time after exposure, latency interval 5 years, $t_{1/2}$ equal to 20 years) results in a lifetime risk of 2.9×10^{-2} if exposure begins at birth and continues at 1 WLM/year.

As discussed in the Section on Health Effects, however, the risk coefficient may increase dramatically at very young ages (less than 10 years) if relative risk methodologies are used to estimate risk coefficients from the atomic bomb survivor data. The results of calculations that used this methodology and assumed (conservatively) that the relative risk coefficient increases over that found in adults by a factor of five (this applies to the 0 to 15 age group) have been reported in the Health Effects Section of the present report. These calculations also assumed a latency interval of five years and no decrease in the relative risk coefficient during the plateau period. Calculated lifetime risk is 6.6×10^{-2}, which is a factor of 2.2 above that calculated by the absolute risk approach detailed above for lifetime exposures to radon progeny at a level of 1 WLM/year. It is likely that the correct value lies within the bounds imposed by the two approaches, but no further analysis of the uncertainty introduced by a lack of clear choice between modeling approaches is possible at present.

This concludes the analysis of uncertainty for lung cancer induction following transfer of radon from water to air. Uncertainties also exist in the doses and risks imposed by direct ingestion of radon-laden water. These uncertainties can be large due to the lack of firmly established metabolic models relating radon ingestion and concentrations in specific body tissues. Fortunately, calculations developed or reported in the present document have employed very conservative assumptions, resulting in what are certainly over-estimates of tissue doses. In addition, these calculations employed "upper bound" estimates of risk coefficients for the G.I. tract. Even with these multiple sources of conservative bias, the risk estimates for the ingestion pathway are small compared to those for the inhalation pathway. As a result, it is likely that complete knowledge of the risks associated with ingestion of radon would not change the total calculated risk from the use of radon-laden water by more than 25% to 50% over that obtained by looking only at the inhalation pathway. Since the risk coefficient assumed for stomach cancer by Cross et al. (1985) is the maximum value suggested by the ICRP (which states that the correct value lies somewhere below this maximum value), it appears certain that incorporation of calculations of the risk from ingested radon will not change the total risk appreciably. This feature negates the need for a complete uncertainty analysis for risk calculations performed for cases of radon ingestion.

In summary, consideration of the potential increase in risk coefficient (risk per WLM) at low levels of exposure indicates that current risk estimates using miner data may be under-estimated by as much as a factor of three to five. The existence of a dose-response threshold above environmental levels would negate this concern entirely. Latency issues could result in uncertainties in lifetime risk that are on the order of 30% or less, while uncertainties in plateau periods impose uncertainties in lifetime risk that are on the order of a factor of three to five. Reasonable lower and upper bound estimates of lifetime risk from uniform exposure to 1 WLM/year over a lifetime differ by a factor of 2.2. Due to a complete lack of empirically derived probability distributions for the various modeling parameters appearing in calculations of lifetime risk, no associated (statistical) level of confidence can be assigned to the statement that the "correct" lifetime risk lies between these upper and lower bounds. This problem arises primarily because of the lack of meaningful measures of the probability of a theoretical approach (such as relative or absolute risk modeling) being correct or incorrect.

When faced with two different conceptual approaches to a problem, the best that can be accomplished currently is to draw inferences using both approaches and then employ those